A RANDOMISED CONTROL STUDY OF EFFECTS OF VARIOUS DOSES OF DOPAMINE CONTINUOUS INTRAVENOUS INFUSION ON THE INTRAOPERATIVE HEMODYNAMICS IN PATIENTS UNDERGOING ELECTIVE SURGERIES UNDER SPINAL ANAESTHESIA

Dissertation submitted in partial fulfilment of the requirements for award of the degree M.D. (Anaesthesiology) Branch X

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CERTIFICATE

This is to certify that this dissertation entitled ***"A RANDOMISED CONTROL STUDY OF EFFECTS OF VARIOUS DOSES OF DOPAMINE CONTINUOUS INTRAVENOUS INFUSION ON THE INTRAOPERATIVE HEMODYNAMICS IN PATIENTS UNDERGOING ELECTIVE SURGERIES UNDER SPINAL ANAESTHESIA"*** submitted by **Dr.SARAVANAN.J** in partial fulfillment for the award of the degree Doctor of Medicine in Anaesthesiology by **The Tamilnadu Dr.M.G.R. Medical University,Chennai** is a bonafide work done by him at **GOVERNMENT KILPAUK MEDICAL COLLEGE, CHENNAI** during the academic year 2013-2016.

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DECLARATION

I, **Dr. SARAVANAN.J**, solemnly declare that this dissertation, entitled "***A RANDOMISED CONTROL STUDY OF EFFECTS OF VARIOUS DOSES OF DOPAMINE CONTINUOUS INTRAVENOUS INFUSION ON THE INTRAOPERATIVE HEMODYNAMICS IN PATIENTS UNDERGOING ELECTIVE SURGERIES UNDER SPINAL ANAESTHESIA***", has been prepared by me, under the expert guidance and supervision of **Prof.Dr.T.Murugan,M.D.,D.A**, Professor and HOD, Department of Anaesthesiology, Government Kilpauk Medical College and Hospital, Chennai and submitted in partial fulfillment of the regulations for the award of the degree **M.D.(Anaesthesiology)** by **The Tamil Nadu Dr. M.G.R. Medical University** and the examination to be held in April 2016.

This study was conducted at Government Kilpauk Medical College Hospital and Government Royapettah Hospital, Chennai. I have not submitted this dissertation previously to any university for the award of any degree or diploma.

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This is to certify that this dissertation entitled ***"A RANDOMISED CONTROL STUDY OF EFFECTS OF VARIOUS DOSES OF DOPAMINE CONTINUOUS INTRAVENOUS INFUSION ON THE INTRAOPERATIVE HEMODYNAMICS IN PATIENTS UNDERGOING ELECTIVE SURGERIES UNDER SPINAL ANAESTHESIA"*** submitted by **Dr.SARAVANAN.J** in partial fulfillment for the award of the degree Doctor of Medicine in Anaesthesiology by **The Tamilnadu Dr.M.G.R. Medical University, Chennai** is a bonafide work done by him at **GOVERNMENT KILPAUK MEDICAL COLLEGE, CHENNAI** during the academic year 2013-2016, under my guidance and supervision.

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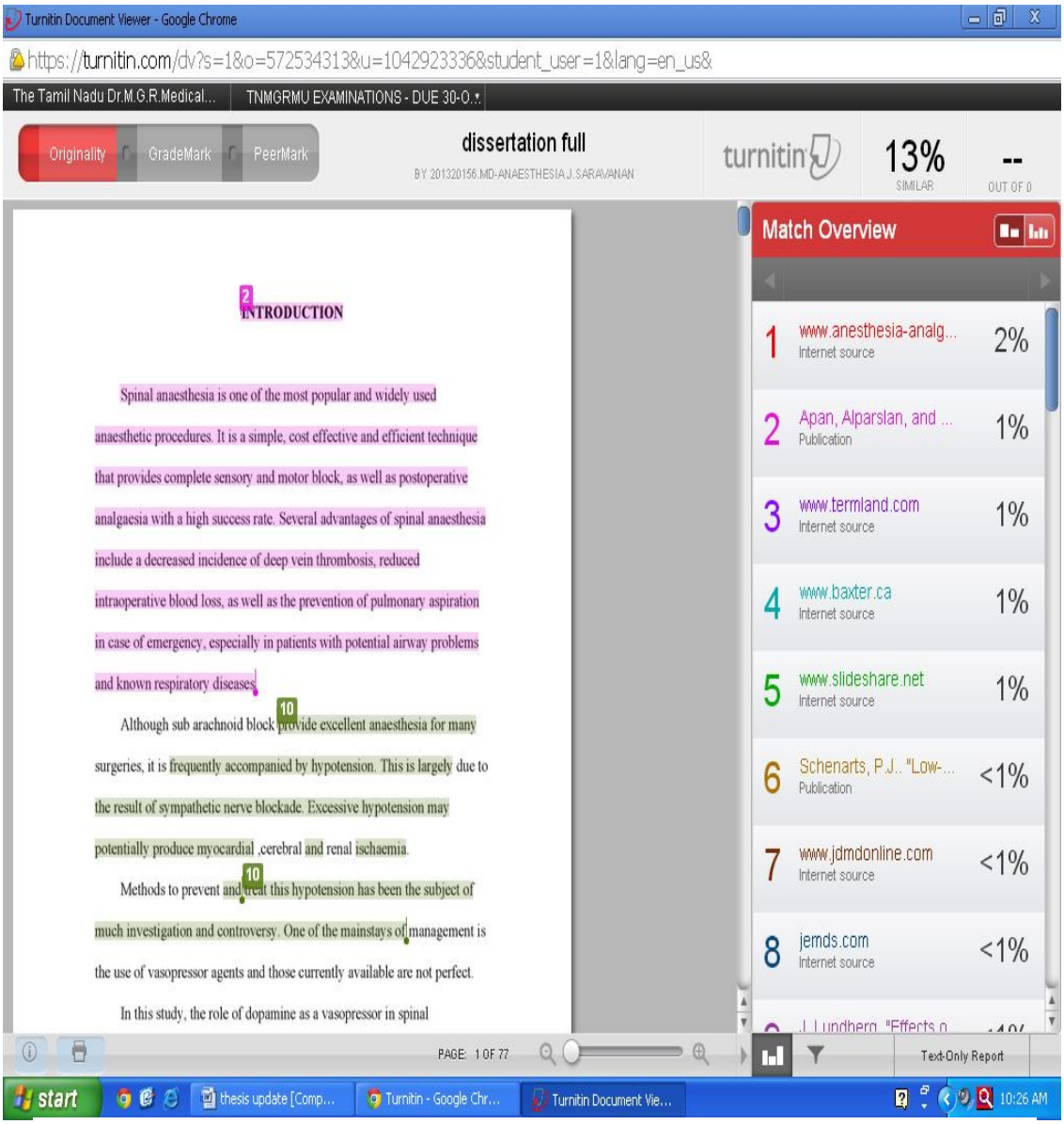
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INTRODUCTION
Spinal anaesthesia is one of the most popular and widely used anaesthetic procedures. It is a simple, cost effective and efficient technique that provides complete sensory and motor block, as well as postoperative analgaesia with a high success rate. Several advantages of spinal anaesthesia include a decreased incidence of deep vein thrombosis, reduced intraoperative blood loss, as well as the prevention of pulmonary aspiration in case of emergency, especially in patients with potential airway problems and known respiratory diseases.
Although sub arachnoid block provide excellent anaesthesia for many surgeries, it is frequently accompanied by hypotension. This is largely due to the result of sympathetic nerve blockade. Excessive hypotension may potentially produce myocardial ,cerebral and renal ischaemia.
Methods to prevent and treat this hypotension has been the subject of much investigation and controversy. One of the mainstays of management is the use of vasopressor agents and those currently available are not perfect.
In this study, the role of dopamine as a vasopressor in spinal
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In this study, the role of dopamine as a vasopressor to spinal hypotension is being studied. Ephedrine was the first agent used for this purpose and it has withstood the test of time, it is the agent against which all

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ABSTRACT AND KEY WORDS

Background: Hypotension is the most common complication during spinal anesthesia. This study aimed to investigate the effects of dopamine on the intraoperative hemodynamics in patients undergoing surgeries under spinal anesthesia.

Material/Methods: This is a randomised control study included 120 patients undergoing elective surgeries under spinal anesthesia. Patients were randomly assigned into 4 groups (n=30 per group):Group A, GroupB, Group C and Group D to receive intravenous dopamine infusion @ 0, 3, 5, and 7 mcg/kg/minute respectively . Pulse rate, blood pressure,mean arterial pressure, ECG, SpO2 were recorded at varying intervals[T1(1^{st} minute),T2(2^{nd} minute),T3,T4,T5]then every 5 minutes upto 30 minutes, then every 10 minutes till the end of the surgery. Urine output was measured every 60 minutes.

Results: When systolic blood pressure was compared between group A and the other 3 groups,there was significant statistical difference between group A and C, A and D,from T2 to T90 minutes, and mean arterial pressure of the three groups B,C and D,compared with control group A,there was statistically significant difference seen between the groups A and C(T2-T100),A and D(T3,T10-T40).Diastolic blood pressure of the control group was compared with the other three groups B,C and D,there was no significant difference statistically.The heart rate of group A is compared with other three groups there was no consistent statistical difference till T30 in group A Vs C, A Vs D,but after T30 minutes there was significant fall in the heart rate in group A when compared to other 3 groups.There was a significant

difference in urine output noted between the control group A and groups which received dopamine infusion.The average volume of urine in control group is 73.9 ml at the end of 60 minutes,but it was around 10 times more in group B , C and D.

Conclusions: Continuous intravenous infusion of 5µg/kg/min dopamine is safe and effective in maintaining hemodynamic stability in patients undergoing surgeries under spinal anaesthesia.

Keywords: Anesthesia,spinal,Hemodynamics,dopamine

INTRODUCTION

Spinal anaesthesia is one of the most popular and widely used anaesthetic procedures. It is a simple, cost effective and efficient technique that provides complete sensory and motor block, as well as postoperative analgaesia with a high success rate. Several advantages of spinal anaesthesia include a decreased incidence of deep vein thrombosis, reduced intraoperative blood loss, as well as the prevention of pulmonary aspiration in case of emergency, especially in patients with potential airway problems and known respiratory diseases.

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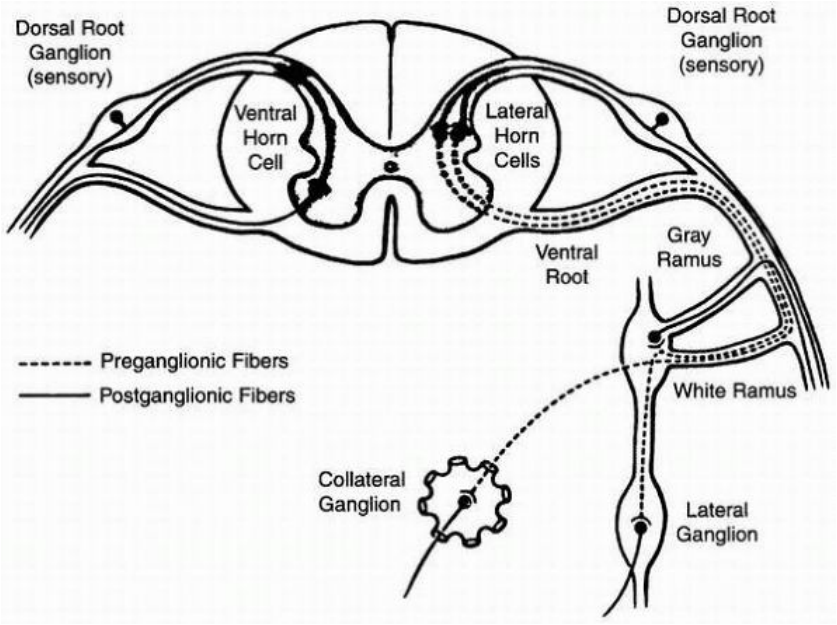
Methods to prevent and treat this hypotension has been the subject of much investigation and controversy. One of the mainstays of management is the use of vasopressor agents and those currently available are not perfect.

In this study, the role of dopamine as a vasopressor in spinal hypotension is being studied. Ephedrine was the first agent used for this purpose and it has withstood the test of time,it is the agent against which all others are compared. It remains the first-line agent in spinal anaesthesia induced hypotension, but it cannot be relied upon to be 100% successful and other agents must be considered when it is inadequate.

Hence in this study the effects of various doses of dopamine continuous intravenous infusion on the intraoperative hemodynamics in patients undergoing elective surgeries under spinal anaesthesia was studied to decide about the appropriate dose of dopamine needed to prevent hypotension in patients undergoing surgeries under spinal anaesthesia.

Physiology of spinal anaesthesia

It is important to understand the alterations in physiology produced by sub-arachnoid blockade in order to make rational choices in managing the resulting hypotension. Spinal block results in a number of changes in the cardiovascular system which may all contribute to the associated hypotension.Essentially, all of the cardiovascular effects of spinal anaesthesia are mediated by blockade of the preganglionic sympathetic neurones produced when local anaesthetic is injected into the subarachnoid space.



1) Reduction in vasomotor tone

This results in arterial and arteriolar vasodilatation, as a direct result of the sympathetic neurone blockade, producing hypotension[1]. Compensatory vasoconstriction may occur in the upper part of the body above the block, as a result of baroreceptor activity[2]. Consequently, those areas with intact sympathetic innervation may be shown to be vasoconstricted as demonstrated by a decrease in skin temperature ,and a decrease in forearm blood flow. As the upper arm blood flow is less than 5% of the cardiac output, the ability to compensate for vasodilatation in the lower body is limited[3].

a)Arterial Circulation (Afterload)

Afterload is the measure of resistance against which the left ventricle must eject blood. It may be measured as the stress (or tension) that is developed in the ventricular wall during systole. Neuraxial anesthesia decreases afterload by producing arterial vasodilation.This vasodilatation, however, is not equivalent in all vascular beds. For instance, muscle and skin blood flow may be decreased by sympathectomy, whereas the total blood flow to the same extremity may be more than quadrupled[4,5].

Additionally, the extent to which afterload is decreased by sympathetic denervation varies considerably from one patient to

another. Therefore, patients with equivalent sympathetic denervation do not necessarily demonstrate equal changes in afterload. Young and robust patients are able to maintain peripheral resistance better than elderly or cachectic patients. The extent to which vascular tone is maintained in various organs is also variable. It is retained most effectively in renal and splanchnic vasculature, less so in skeletal muscle, and least effectively in skin vessels.

When arterial vasodilation results from sympathetic denervation, a simultaneous, compensatory reflex vasoconstriction occurs in areas of the body in which the sympathetic nervous system is intact, usually in areas cephalad to the site of spinal anesthetic block. The effectiveness of this reflex vasoconstriction in maintaining normotension is a function of the extent of the sympathetic block. If, for instance, sympathetic denervation reaches the fourth thoracic dermatome (T4) or higher, the intact upper limb vasculature may contribute only 5% of the total cardiac output (CO). Even maximal vasoconstriction will be insufficient to compensate for the profound arterial vasodilation in the rest of the body. Reflex cerebral vasoconstriction does not occur, because of the intrinsic cerebral autoregulatory mechanisms.

b)Venous Circulation (Preload)

Preload is a measure of the volume of venous blood returned to the right ventricle from the periphery. Sympathetic denervation associated with central neuraxial block dilates not only the arterial and postarteriolar circulation (afterload), but also the venous circulation. Neural innervation of the venous circulation is similar to that of the arterial circulation, and areas of vasodilation will be equivalent. However, the degree of vasodilation in the arterial and venous sides of the circulation is markedly different. Because the arterial walls contain more smooth muscle fibers and supporting structures (media) than their venous counterparts, sympathetic denervation results in less vasodilation in afterload vessels, because they maintain their own intrinsic vascular tone. In contrast, the preload (venous) vasculature undergoes maximal dilatation, and venous capacitance increases maximally. This change results in the rapid pooling of an abnormally large volume of blood in the periphery and a marked decrease in the blood volume returning to the right ventricle, resulting in a significant decrease in CO and a decrease in BP.

2) Loss of cardio accelerator nerve function

Blockade above the T5 level results in the removal of the chronotropic and inotropic influence of the sympathetic nervous system.Peripheral venodilation also diminishes venous return, which contributes to the bradycardia. Cardiac output, and hence blood pressure, may therefore fall[6,7].

i)Heart Rate

Slowing of the HR is characteristically associated with neuraxial anesthesia, and the extent of bradycardia correlates well with the extent of sympathetic denervation. However, the relation between denervation and the degree of bradycardia may be modified by a number of other factors including age, coadministration of intravenous drugs, and the position of the patient on the operating room table. Importantly, bradycardia during high (thoracic) levels of spinal anesthesia is due to two main factors:

(i) denervation of preganglionic cardiac accelerator fibers (T1-T4)

(ii) diminished venous return to the right ventricle because of decreases in preload.

In extreme cases, the rapid decrease in venous return to the right ventricle (due to massive hemorrhage or assumption of head-up positioning during spinal anesthetic-induced near-total sympathectomy) may result in activation of mechanoreceptors and chemoreceptors in the ventricle. This activation results in severe bradycardia or asystole due to further increases in parasympathetic activity and inhibition of sympathetic activity (Bezold-Jarisch reflex induced asystole).

ii)Cardiac Output

Reduced Cardiac Output following spinal anesthesia is one of the most consistent findings reported in the literature and is the *sine qua non* of neuraxial anesthesia. The extent of Cardiac Output decrease also is a function of the degree of sympathetic denervation. Because one of the many determinants of Cardiac Output is the amount of blood in the ventricle (preload), and preload is exquisitely sensitive to the effects of gravity, marked changes in Cardiac Output may be induced by patient positioning. Placing patients undergoing neuraxial anesthesia in the horizontal position while elevating the legs will facilitate venous return to the heart and tends to maintain Cardiac Output and BP. Conversely, assumption of an even slight head-up position during neuraxial anesthesia with high levels of sympathetic

denervation (in the misguided attempt to prevent further extension of the spinal block) may have catastrophic consequences such as profound bradycardia, cerebral hypoperfusion, and cardiac arrest.Reports of severe complications related to improper positioning of patients (i.e., head-up) during high levels of spinal anesthesia have spanned the last six decades[8,9].

iii)Myocardial Work[10]

Because high levels of sympathetic denervation are associated with decreased afterload (and preload), the amount of work performed by the heart er unit time is decreased. Eckenhoff demonstrated in dogs with a spinal-induced total sympathectomy, a 66% decrease in left ventricular work.The significant decrease in myocardial work is due primarily to three factors:

(i) decrease in heart rate

(ii) decrease in arterial/total peripheral resistance (afterload)

(iii) decrease in stroke volume of the left ventricle secondary to the decreased preload.

iv)Myocardial Irritability[11]

It would seem unlikely that myocardial irritability should be increased during high levels of sympathetic denervation produced by

neuraxial anesthesia. Indeed, the development of tachyarrhythmia has not been reported. There are, however, reports of sinus bradycardia and even asystole in patients with sick sinus syndrome undergoing spinal anesthesia.

v)Coronary Perfusion[12,13,14]

Perfusion is determined by the difference between the driving force (mean aortic pressure) and the coronary vascular resistance. The sympathectomy-induced decrease in mean aortic pressure does not have a deleterious effect on coronary perfusion because of coronary circulation autoregulation. The decrease in coronary perfusion pressure is compensated by several factors:

(i) decreased ventricular intramural pressure during diastole, when coronary perfusion takes place;

(ii) decreased rate of contraction of the ventricle, which decreases myocardial oxygen demand;

(iii) coronary autoregulation.

Hackel et al reported that in patients with high spinal anesthetic-sympathetic denervation, the mean arterial pressure decrease (48%) was compensated by a relatively larger decrease in myocardial work and oxygen utilization (53%).

Neuraxial anesthesia also induces favorable changes in the distribution of coronary blood flow. Following myocardial infarction, sympathetic blockade associated with high thoracic epidural anesthesia increases subendocardial perfusion more than epicardial flow by decreasing left ventricular end-diastolic pressure and left ventricular wall tension.Similar beneficial effects of thoracic epidural anesthesia were reported in patients with unstable angina pectoris.

3)Systemic uptake of local anaesthetic

Equal doses of local anaesthetic administered into the subarachnoid and epidural spaces produce similar plasma concentrations of drug (75 mg lidocaine produce a plasma concentration of 0.32 + 0.07 mg/ ml after subarachnoid injection versus 0.41 + 0.07 mg/ml after epi- dural injection), but the absorption is more rapid from the epidural space, presumably because of its greater vascularity. The much smaller doses of local anaesthetic used for a spinal block can be expected to have little systemic effect, but the absorption of large doses from the epidural space may be considerable. Local anaesthetics used intravenously for control of cardiac arrhythmias are known to cause myocardial depression and it has been shown that epidural administration of such drugs will also

produce plasma concentrations great enough for systemic effects to be produced (blood lidocaine concentrations have been measured at between 3 and 7 mg/ml. It has been shown that lumbar epidural anaesthesia (without blocking the cardio accelerator fibres) can reduce cardiac output and arterial blood pressure[15,16,17,18].

4)other factors

Neuraxial anesthesia-induced hypotension usually develops in the first 15 to 20 minutes. Many factors influence the time course of hypotension, including:

- Speed of injection
- Total dose and/or volume and concentration of local anesthetic
- Intravascular volume status
- Patient positioning
- Patient comorbidities (hypertension, diabetes and autonomic dysfunction, chronic diuretic or beta-blockade therapy, etc.)

Severe hypotension from spinal anesthesia may be predicted reliably by analyzing HR variability, which is an indirect measure of autonomic control.

If spinal anaesthesia is performed in a standardised manner on a large series of patients, keeping the height of block and position of the patient constant in all cases, some patients will show very little alteration in blood pressure while others will show pronounced hypotension. There may be many reasons for this variabilty in response, such as pregnancy,old age, pre-existing hypertension or cardiovascular disease, and hypovolaemia.

Patients with pre-existing hypertension undergoing spinal anaesthesia show a much greater decrease in blood pressure and peripheral vascular resistance than normotensive patients with equivalent levels of anaesthesia. Age is a major factor in determining the haemodynamic response to spinal anaesthesia, the degree of hypotension increasing with increasing age.The effect of co-existing medical disease on hypotension induced by spinal anaesthesia can also be shown to be of importance. Patients in ASA class III may produce a more pronounced hypotensive response than patients in ASA classes I or II. Spinal anaesthesia in the presence of hypovolaemia may be associated with severe hypotension and cardiovascular depression, and should be avoided in this situation whenever possible[19,20].

MANAGEMENT OF HYPOTENSION IN SPINAL ANAESTHESIA

Hypotension could be a major problem with spinal anaesthesia. The management of this problem therefore became important in order that the quality of anaesthesia produced by these blocks could be matched by safety.

Methods developed for the management of the hypotension fall into four categories:

i Volume expansion

ii Physical methods to increase in venous return.

iii Prevention or treatment of associated bradycardia

iv Vasoconstriction

These techniques have been used separately and in combination with varying degrees of success, and the optimal form of management remains the subject of much discussion.

(i) Volume expansion

It is common practice to infuse large volumes (10-20 ml. kg) of electrolyte crystalloid solution (e.g., saline 0.9%) rapidly to help prevent or to treat hypotension induced by a spinal block[21]. This will increase venous return and therefore cardiac output[22,23].Haemodilution

also Occurs, which will improve peripheral circulation, but this may be at the expense of oxygen delivery[24]. The use of excessive volumes of crystalloid may therefore do more harm than good. Crystalloid preloading may also be relatively ineffectual in preventing hypotension[25].

Although preloading reduced the incidence of hypotension, there was still a considerable proportion of the patients who became hypotensive and the clinical relevance of the reduction in the incidence of hypotension is questionable. A further problem is the increased need for catheterisation as a result of developing urinary retention as a combined effect of the spinal block itself and the fluid load given. Large fluid loads may be poorly tolerated by patients with limited myocardial reserve or with a relatively fixed cardiac output because of valvular heart disease. Some of these problems may be lessened by the use of smaller volumes of colloid solutions, but again this approach may not be completely successful, and colloid solutions have been known to produce anaphylactoid reactions in a small number of patients[26].

It is therefore clear that intravenous fluids do not provide a complete solution to this problem.

(ii) Physical methods to increase venous return

Venous return can be augmented by elevating the patient's legs (providing the femoral veins remain unobstructed) or by the use of a head-down tilt. These manoeuvres alone may be sufficient to restore blood pressure to an acceptable level. However, this is not always the case and other methods must then be used. The head-down position may also be regarded as undesirable when hyperbaric local anaesthetic solutions are used because of concern over cephalad spread of the block[27,28].

(iii) Prevention or treatment of associated bradycardia

Spinal blockade affecting the upper thoracic spinal cord segments (T_1-T_4) produces bradycardia by blocking the cardioaccelerator nerve fibres, allowing vagal tone to dominate.As heart rate is one of the determinants of cardiac output and hence blood pressure, drugs with vagolytic actions (e.g., atropine) can be used to elevate heart rate and hence blood pressure. However, the response is erratic and the disadvantages may outweigh the benefits. In particular, the production of a tachycardia is undesirable in patients dependent on heart filling and coronary perfusion during diastole[29].

(iv) vasoconstriction

Vasopressors are the mainstay in the management of spinal anaesthesia induced hypotension.Understanding the pathophysiology of sympathectomy-induced hypotension is extremely important in the selection of vasopressors for treatment. Cardiac output may be increased by increasing heart rate, increasing stroke volume, or both. Atropine may increase cardiac output through its chronotropic effects on heart rate. However, it is rarely effective by itself during sympathectomy-induced bradycardia and hypotension because of its lack of vasoconstrictive properties. In such instances, drugs that provide both chronotropic and venoconstrictive effects are preferred.

Because severe hypotension and associated bradycardia in high sympathetic denervation result from the marked increase in venous capacitance, vasoactive substances should increase preload preferentially.

A vasoconstrictor that predominantly increases afterload (on a background of low preload) may increase peripheral BP toward normal, but will further decrease perfusion pressure to the core organs because of arterial vasoconstriction. Mixed adrenergic agonists (such as ephedrine,dopamine) correct hypotension more effectively than

either pure alpha-adrenergic (phenylephrine) or beta-adrenergic (isoproterenol) agonists. Ephedrine provides both venoconstriction and chronotropy, thereby reversing the effects of denervation of the T1-4 cardio-accelerator fibers. On the other hand, an agent such as phenylephrine may increase afterload by increasing arterial vasoconstriction and further induce reflex bradycardia. Of the currently available sympathomimetic amines, norephinephrine has the most venoconstrictive properties, followed by metaraminol, ephedrine, mephentermine, and phenylephrine.

History of the use of vasopressor agents:

Ephedrine was the first agent to be used successfully to treat hypotension induced by spinal anaesthesia, in 1927. Later research examined the effects of other drugs, including paredrine, methedrine, pitressinephedrine combination, and later methoxamine, phenylephrine, metaraminol, mephentermine, dopamine and dobutamine.

Dopamine[30]:

Dopamine is an endogenous catecholamine that is the immediate precursor of norepinephrine. Dopamine regulates cardiac, vascular, and endocrine function and is an important neurotransmitter in the central and peripheral nervous systems.

The pharmacology of dopamine is complex as this catecholamine differentially stimulates a variety of dopaminergic as well as adrenergic receptors. It is a relatively nonspecific agonist at both dopamine1 (D1) and dopamine2 (D2) receptors and the α- and β-adrenergic receptors. D1 receptors are located postsynaptically.

Mechanism of action

When activated D1 receptors elicit vasodilation in renal, mesenteric, coronary, and cerebral vascular beds and inhibition of sodium–potassium adenosine triphosphatase .Activation of these receptors is mediated by adenylate cyclase stimulation. D2 receptors are principally presynaptic and inhibit adenylate cyclase activity and release of norepinephrine in autonomic nervous system ganglia and adrenergic nerves (in renal and mesenteric vessels) leading to vasodilation. D2 receptors are also present in the pituitary gland, emetic center of the medulla, and kidney. Nausea and vomiting

produced by dopamine probably reflect stimulation of D2 receptors. Dopamine receptors may also be associated with the neural mechanism for “reward” that is associated with cocaine and alcohol dependence.

Pharmacokinetics

Traditionally, the pharmacokinetics of dopamine has been attributed to dose-dependent effects on varying receptors.At low intravenous (IV) infusion rates (0.5 to 3 μg/kg/minute), dopamine primarily stimulates D1 and D2 receptors leading to vasodilation, decreased arterial blood pressure, and increased renal and splanchnic vascular blood flow. Diuresis and natriuresis also occur. The decrease in diastolic blood pressure might lead to a reflex increase in heart rate.

At higher infusion rates (3 to 10 μg/kg/minute), dopamine primarily stimulates β1-adrenergic receptors in the heart as well as α receptors in the peripheral vasculature. It also induces norepinephrine release from vascular sympathetic neurons. The activation of β receptors leads to increased cardiac output by increasing chronotropy and contractility along with vasodilation and afterload reduction.

As the infusion rate is increased even further (>10 μg/kg/minute), dopamine predominantly stimulates α1 receptors, acting similarly to a

pure α agonist. The predominant stimulation of vascular smooth muscle α1 receptors at these higher doses lead to arterial and venous vasoconstriction, increased systemic vascular resistance, and increased blood pressure attenuating further increases in cardiac output. Reflex bradycardia may also occur at this point.

This aforementioned dose-dependent model of dopamine's effects is too simplistic, even in healthy individuals. There are a wide range of clinical responses depending on individual variability in pharmacokinetics as well as other variables.

For example, despite identical IV infusion rates, there may be a 10- to 75-fold variability in plasma dopamine concentrations produced even in healthy individuals with normal drug metabolism. The etiology of the wide pharmacokinetic variability and variation in individual responses is likely multifactorial, reflecting differences in drug distribution, elimination, and endogenous levels, among other factors. Such differences may be even more profound in critically ill patients. Hence, the effects of dopamine cannot be predicted based on the dose, and the drug must be titrated to effect.

Dopamine increases cardiac output by stimulation of β1 receptors, increasing stroke volume. This increase in cardiac output is usually

accompanied by only modest increases in heart rate, systemic blood pressure, and systemic vascular resistance. A portion of the effect of dopamine is also due to stimulation of endogenous norepinephrine release, which may predispose to the development of cardiac dysrhythmias. Nevertheless, dopamine is less dysrhythmogenic than epinephrine. The release of norepinephrine caused by dopamine may be an unreliable mechanism for increasing cardiac output when catecholamine stores are depleted, as occurs with patients in chronic cardiac failure.

Dopamine increases myocardial oxygen consumption.Dopamine causes both relaxation and contraction of vascular smooth muscle with the predominant effect varying by vascular bed, predominant receptor type, and dose administered. Dopamine's effect on pulmonary vascular resistance has not been well studied in humans, though there are some reports it may decrease pulmonary vascular resistance in patients with chronic obstructive pulmonary disease.

Rapid metabolism of dopamine with an elimination half-life of 1 to 2 minutes mandates its use as a continuous infusion (1 to 20 µg/kg/minute) to maintain therapeutic plasma concentrations. Dopamine should be dissolved in 5% glucose in water for IV administration to avoid the inactivation that may occur in alkaline

solutions. Extravasation of dopamine, like norepinephrine, produces intense local vasoconstriction, which may be treated with local infiltration of phentolamine.

Dopamine is not effective orally and does not cross the blood–brain barrier in sufficient amounts to cause CNS effects. The immediate precursor of dopamine, L-dopa, is absorbed from the gastrointestinal tract and readily crosses the blood–brain barrier. Dopamine is partially protein bound. Approximately 25% is converted to norepinephrine. Dopamine undergoes metabolism in the liver with conjugation to sulfates and glucuronides, pulmonary endothelium by catechol-O-methyltransferase (COMT) and excretion by the kidneys.

Clinical Uses

Dopamine is used clinically to increase cardiac output in patients with decreased contractility, low systemic blood pressure, and low urine output as may be present after cardiopulmonary bypass or with chronic heart failure. It is unique among the catecholamines in being able to simultaneously increase myocardial contractility, renal blood flow, glomerular filtration rate, excretion of sodium and urine output.

Dopamine exerts its positive chronotropic, dromotropic, inotropic, and lusitropic (myocardial relaxant) effects via β1-adrenergic

receptors. Activation of arterial and venous α1 receptors increases systemic vascular resistance, preload, and left ventricular afterload.However, as dopamine increases pulmonary vascular resistance as well, it may not be the preferred inotropic agents in patients with pulmonary hypertension or right ventricular dysfunction.

Renal-Dose Dopamine

The term renal-dose dopamine or low-dose dopamine refers to the continuous infusion of small doses (1 to 3 μg/kg/minute) of dopamine to patients to promote renal blood flow. In healthy individuals, low-dose dopamine increases renal blood flow and induces natriuresis and diuresis Theoretically, dopamine's renal vasodilating effects may be useful in patients with impaired renal function or in patients at risk of decreased renal perfusion as may occur with decreased cardiac output.

Small doses of dopamine increase renal blood flow predominantly by D1 receptors in the renal vasculature and possibly by D2 receptors via inhibition of norepinephrine release. Larger doses predominantly increase renal blood flow by β-adrenergic–mediated increases in cardiac output. Higher doses of dopamine presumably stimulate α receptors to increase perfusion pressure. In addition, dopamine

triggers natriuresis and diuresis through direct effect on tubular cell function. Dopamine binds to D1 and D2 receptors in the proximal tubule, thick ascending loop of Henle, and cortical collecting ducts inhibiting sodium-potassium ATPase activity, increasing sodium excretion, inducing natriuresis and diuresis.

The activation of D2 in inner medullary collecting ducts stimulates prostaglandin E2 production. This antagonizes the effects of antidiuretic hormone and results in increased free water clearance. PGE2 enhances blood flow in the inner medulla. Inhibition of aldosterone also increases sodium excretion and diuresis. Hence, dopamine has direct and indirect effects on the renal vasculature in addition to functioning as a diuretic.

The term renal-dose or low-dose dopamine is misleading as dopamine has many effects at sites other than the kidneys, even at low doses. Dopamine’s effects based on dose alone are unpredictable. Low-dose dopamine also implies an unproven beneficial effect on renal function.In patients receiving dopamine before a “renal insult,” there is a clear diuretic effect but none for improved creatinine clearance or decreased need for hemodialysis.Despite drug-induced diuresis, there is no evidence that urine output in the presence of low cardiac output and/or hypovolemia protects renal function. The use of

dopamine after the renal insult has occurred has not been shown to improve glomerular filtration rate. There is evidence that the beneficial effect of low-dose dopamine on renal blood flow and glomerular filtration rate observed in healthy individuals is due to drug-induced increases in cardiac output, and this benefit is lost in early renal failure.

No randomized controlled studies have demonstrated a decrease in the incidence of acute renal failure when dopamine is administered to patients considered to be at risk for developing acute renal failure in multiple patient populations (major vascular surgery, cardiopulmonary bypass, intensive care, heart failure, sepsis, transplantation, patients exposed to nephrotoxic drugs) confirming the results of two large retrospective studies and two meta-analyses, finding that dopamine does not prevent or reverse acute renal failure or improve outcome. Aside from one study demonstrating dopamine selectively increases renal blood flow in heart failure patients via dilation of both large conductance and small resistance renal blood vessels, there exists no proven improvement in renal perfusion, creatinine clearance, or glomerular filtration rate and no alteration in the course of renal failure or the need for renal replacement therapy.

Dopamine leads to an increase in cortical and inner medullary blood flow, shunting flow from the outer medulla. It increases solute delivery to the distal tubular cells increasing medullary oxygen consumption. The outer medulla is highly metabolically active and highly

susceptible to ischemic injury and acute renal failure. Dopamine can induce renal failure in both normo- and hypovolemic patients.

Low-dose dopamine is associated with multiple complications affecting the cardiovascular, pulmonary, gastrointestinal, endocrine, and immune systems.

Cardiovascular Effects

Dopamine is associated more than dobutamine or epinephrine with dose-related sinus tachycardia and the potential to cause ventricular arrhythmias and may predispose to myocardial ischemia by precipitating tachycardia, increasing contractility, increasing afterload, and precipitating coronary artery vasospasm. Dopamine increases peripheral vascular resistance and pulmonary artery pressures. Unlike dobutamine, dopamine does not inhibit hypoxic pulmonary vasoconstriction. Nevertheless, dopamine is not recommended for use in right heart failure.

Gastrointestinal Effects

Gastrointestinal mucosal ischemia and subsequent translocation of bacteria and bacterial toxins play an important role in the development of multiple organ dysfunction syndrome. Dopamine's effect on splanchnic blood flow and gastric intramucosal pH is controversial and inconsistent. There is no evidence that low-dose dopamine has beneficial effects on splanchnic function or reduces the progression to multiorgan failure in sepsis. Dopamine may increase flow to the muscular layer of the gut with decreased flow to the mucosal layer with detrimental effects and possibly gut ischemia. In septic patients, dopamine but not norepinephrine, as administered to maintain an acceptable mean arterial pressure, resulted in an uncompensated increase in splanchnic oxygen requirements. Although dopamine infusion in septic patients led to increased hepatosplanchnic perfusion, hepatosplanchnic oxygen uptake was reduced suggesting an impairment of hepatosplanchnic metabolism.Most of these studies have looked at the effects of low-dose dopamine rather than doses used to treat hypotension.

D2 receptors are also located in the enteric nervous system. Dopamine agonists interfere with gastrointestinal motility. Low-dose

dopamine has been demonstrated to slow gastric motility in mechanically ventilated intensive care patients.

Endocrine and Immunologic Effects

Dopamine disrupts metabolic and immunologic functions through its effects on hormones and lymphocyte function. The anterior pituitary plays a crucial role in metabolic and immunologic homeostasis. The initial stress response stimulates pituitary hormone release, whereas the chronic phase is associated with suppression of the hypothalamic-pituitary axis. In the acute phase of an illness, dopamine induces the pattern of hypopituitarism seen in prolonged critical illness and chronic stress. When dopamine is used in the chronic phase of illness, it further suppresses the circulating concentrations of pituitary hormones.

Dopamine depresses the immune status by reducing serum prolactin levels. Prolactin is an immunoregulatory hormone affecting T and B lymphocytes. Dopamine inhibits lymphocyte proliferation, immunoglobulin synthesis, and cytokine production and promotes lymphocyte apoptosis. Dopamine also decreases the secretion of growth hormone, which has anabolic, lipolytic, and immune-stimulating properties. Growth hormone deficiency can

contribute to impaired anabolism and a negative nitrogen balance. Dopamine's inhibition of thyrotropin-releasing hormone leads to "euthyroid sick syndrome." It also decreases dehydro-epiandrosterone sulfate and may affect luteinizing hormone release. Dopamine's overall effect is to suppress the secretion and function of anterior pituitary hormones, aggravating catabolism and cellular immune function and inducing central hypothyroidism.

Respiratory Effects

The infusion of low-dose dopamine in healthy subjects as well as heart failure patients interferes with the ventilatory response to arterial hypoxemia and hypercapnia, reflecting the role of dopamine as an inhibitory neurotransmitter at the carotid bodies.The result is depression of ventilation in patients who are being treated with dopamine to increase myocardial contractility. Dopamine also decreases arterial oxygen saturation by impairing regional ventilation/perfusion matching in the lungs. Dose-dependent reductions in arterial PO_2 with increasing rates of dopamine in critically ill patients after major surgery have been demonstrated. Arterial blood gases have been observed to deteriorate during infusion of dopamine.

One study demonstrated low-dose dopamine did not influence ventilation specifically in patients with chronic obstructive pulmonary disease either breathing spontaneously or being weaned from mechanical ventilation. Dopamine may even decrease pulmonary vascular resistance in patients with chronic obstructive pulmonary disease. Other potential beneficial effects are improved respiratory muscle contraction, increased lung edema clearance, and inhibition of bronchoconstriction. However, such reports are anecdotal or not tested in clinical studies.

Intraocular Pressure

Continuous infusions of dopamine to critically ill patients are associated with increases in intraocular pressure.This may create a risk in patients with preexisting glaucoma especially if they are sedated and mechanically ventilated.

PHARMACEUTICAL INFORMATION

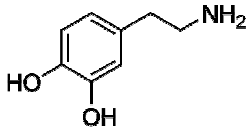
Proper name: Dopamine Hydrochloride

Chemical name: 3,4-dihydroxyphenethylamine hydrochloride

Molecular formula: $C_8H_{11}NO_2$

Molecular mass: 189.64

Structural formula:

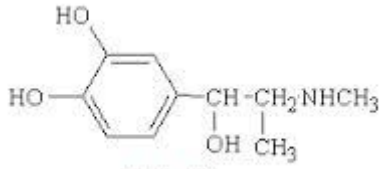


Physicochemical properties: Dopamine hydrochloride is a white, odorless, crystalline powder, soluble in water and alcohol with a melting point range of 240° to 248°C (with decomposition). It is sensitive to light, alkalis, iron salts and oxidizing agents.

Synthetic Noncatecholamines

The commonly used noncatecholamine sympathomimetic drugs are ephedrine and phenylephrine.

Ephedrine[31]



Ephedrine

Ephedrine is an indirect-acting synthetic sympathomimetic that stimulates α- and β-adrenergic receptors. It is the active principle of the Chinese plant Ma Huang, used for centuries in the East. It was first brought to Europe in 1923 and it is now produced synthetically.The pharmacologic effects of ephedrine are partly due to direct stimulation of adrenergic receptors (direct-acting) and partly due to stimulation of release of endogenous norepinephrine (indirect-acting). Ephedrine is resistant to metabolism by monoamine oxidase (MAO) in the gastrointestinal tract, thus permitting unchanged drug to be absorbed into the circulation after oral administration. Structurally it has two asymmetrical carbon atoms: only l-ephedrine and racemic ephedrine are used clinically.Intramuscular injection of ephedrine is clinically

acceptable because drug-induced local vasoconstriction is insufficient to delay systemic absorption or lead to tissue injury.

Up to 40% of a single dose of ephedrine is excreted unchanged in urine. Some ephedrine is deaminated by MAO in the liver, and hepatic conjugation also occurs. The slow inactivation and excretion of ephedrine are responsible for the prolonged duration of action of this sympathomimetic.

Ephedrine, unlike epinephrine, does not produce marked hyperglycemia. Mydriasis accompanies the administration of ephedrine, and CNS stimulation does occur, although less than that produced by amphetamine.Myocardial irritability is increased, which may produce arrhythmias. Other actions include a reduction in cerebral and renal blood flow, bronchodilation (hence its use in treating asthma), increase in sphincter tone (which may lead to urinary retention).

Clinical Uses

Ephedrine, 5 to 10 mg IV administered to adults, is a commonly selected sympathomimetic to increase systemic blood pressure in the presence of sympathetic nervous system blockade produced by regional anesthesia or hypotension due to inhaled or injected

anesthetics. In an animal model, ephedrine more specifically corrected the noncardiac circulatory changes produced by spinal anesthesia than did a selective α or β agonist drug.

Until recently, ephedrine was considered the preferred sympathomimetic for administration to parturients experiencing decreased systemic blood pressure owing to spinal or epidural anesthesia. Support for this practice was the observation in pregnant ewes that uterine blood flow was not greatly altered when ephedrine was administered to restore maternal blood pressure to normal after production of sympathetic nervous system blockade. Recent reviews of trials of ephedrine versus phenylephrine have concluded that systemic blood pressure control is similar with both drugs but phenylephrine is associated with a higher umbilical artery pH at delivery than ephedrine.

Administration of phenylephrine by infusion during cesarean section to maintain maternal systolic blood pressure at baseline is associated with a lower incidence of fetal acidosis than is ephedrine. Based on these data, it seems that α agonists such as phenylephrine may be preferable to ephedrine for treatment of maternal hypotension.

Ephedrine can be used as chronic oral medication to treat bronchial asthma because of its bronchodilating effects by activation of β2-adrenergic receptors. Compared with epinephrine, the onset of action of ephedrine is slow, becoming complete only 1 hour or more after administration. A decongestant effect accompanying oral administration of ephedrine produces symptomatic relief from acute coryza. Ephedrine, 0.5 mg/kg intramuscularly, has an antiemetic effect similar to that of droperidol but with less sedation when administered to patients undergoing outpatient laparoscopy using general anesthesia.

Cardiovascular Effects

The cardiovascular effects of ephedrine resemble those of epinephrine, but its systemic blood pressure–elevating response is less intense and lasts approximately 10 times longer. Intravenous ephedrine results in increases in systolic and diastolic blood pressure, heart rate, and cardiac output. Renal and splanchnic blood flows are decreased, whereas coronary and skeletal muscle blood flows are increased. Systemic vascular resistance may be altered minimally because vasoconstriction in some vascular beds is offset by vasodilation (β2 stimulation) in other areas. These cardiovascular

effects are due, in part, to α receptor–mediated peripheral arterial and venous vasoconstriction.

The principal mechanism, however, for cardiovascular effects produced by ephedrine is increased myocardial contractility due to activation of β1 receptors. In the presence of preexisting β-adrenergic blockade, the cardiovascular effects of ephedrine may resemble responses more typical of α-adrenergic receptor stimulation.

A second dose of ephedrine produces a less intense systemic blood pressure response than the first dose. This phenomenon, known as tachyphylaxis, occurs with many sympathomimetics. Tachyphylaxis to ephedrine appears to involve α receptor inhibition.

REVIEW OF LITERATURE

1. **Cabalum et al 1979**[32] studied the Effects of dopamine on hypotension induced by spinal anesthesia and found that chronically instrumented, near-term pregnant sheep were subjected to autonomic blockade with spinal anesthesia. Systemic arterial pressure, heart rate, and uterine blood flow decreased and uterine vascular resistance increased during the spinal blockade. Infusion of dopamine during the spinal hypotension corrected the disturbed circulatory parameters. Dopamine represents a useful agent in the management of spinal hypotension.

2. **Clark RB et al** [33]found that Dopamine has been shown to decrease uterine blood flow in animals, and has not been shown to have any benefit over ephedrine in humans.

3. **Butterworth et al**[34] found that acute denervation of spinal anesthesia altered venous and arterial dose-response relationships of both dopamine and dobutamine. Their study demonstrates the effectiveness of dobutamine and, perhaps even more so, dopamine as possible alternatives to ephedrine for the pharmacologic correction of the non cardiac circulatory sequelae of spinal anesthesia.

4. **Lundberg et al**[35] found that myocardial contractility and arterial pressure were restored to pre-Thoracic Epidural Analgesia values by dopamine at 5–10 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

5. **Toshiharu Kasaba et al**[36]found that Dopamine is preferable to ephedrine and dobutamine in providing hemodynamic stability during propofol induction and tracheal intubation following epidural anesthesia.

6. **J.F.Lundberg et al**[37] found that the systemic pressure response induced by dopamine was augmented during epidural anesthesia. Norepinephrine did not increase arterial pressure and myocardial contractility as markedly as dopamine, and cardiac output was not altered.

7. **Rolbin SH et al**[38] found that treatment of spinal hypotension with dopamine decreases uterine blood flow in the pregnant ewe/animals, and has not been shown to have any benefit over ephedrine in humans.

8. **Defeng Sun et al**[39] found that dopamine can be used to correct epidural anesthesia-induced hypotension and compared the effects of methoxamine versus dopamine on the hemodynamic parameters.Their findings suggest that methoxamine can have an

effect similar to that of dopamine in controlling hemodynamic stability, but it is superior to dopamine in reducing the risk of a tachycardia-induced increase in myocardial oxygen consumption in elderly patients.

9. ***Ward RJ, Kennedy WF Jr, Bonica JJ, Martin WE, Tolas AG, Akamatsu T*.et al**[40] Did experimental evaluation of atropine and vasopressors for the treatment of hypotension of high subarachnoid anesthesia and found that atropine is not useful for spinal hypotension and ephedrine is a better option for spinal hypotension.

10. ***Butterworth JF IV, Piccione W Jr, Berrizbeitia LD, Dance G, Shemin RJ, Cohn LH*.et al**[41] Studied on augmentation of venous return of leg using adrenergic agonists during spinal anesthesia and concluded that a mixed adrenergic agonist such as ephedrine more ideally corrects the noncardiac circulatory sequelae of spinal anesthesia than does either a pure [alpha]-or [beta]-adrenergic agonist.

11. **Nygan Kee WD, Khaw KS, Ng FF, Lee BB.et al**[42]Did a study on Prophylactic phenylephrine infusion for preventing hypotension during spinal anesthesia for cesarean delivery and found that

prophylactic phenylephrine infusion is a simple, safe, and effective method of maintaining arterial blood pressure during spinal anesthesia for cesarean delivery.

12. **Frazer RS Edwards GM. et al**[43]Did a study using prophylactic ephedrine infusion in obstetric anaesthesia and concluded that prophylactic ephedrine should be used in obstetric as well as in other cases of spinal anaesthesia to prevent hypotension.

13. **Kang YG, Abouleish E, Caritis S. et al**[44]Studied about prophylactic intravenous ephedrine infusion during spinal anesthesia for cesarean section and The results suggest that prophylactic ephedrine infusion is safe and desirable in healthy parturients undergoing cesarean section under spinal anesthesia.

14. ***Gajraj NM, Victory RA, Pace NA, Van Elstraete AC, Wallace DH*.et al**[45] Did a comparison of an ephedrine infusion with crystalloid administration for prevention of hypotension during spinal anesthesia and concluded that a prophylactic ephedrine infusion is effective for minimizing and managing hypotension associated with spinal anesthesia and compares favorably with crystalloid administration in this patient population in terms of efficacy and incidence of side effects.

15. ***Hemmingsen C, Poulsen JA, Risbo A*. Et al**[46]Did a double-blind study using prophylactic ephedrine during spinal anaesthesia in patients in ASA groups I–III and concluded that prophylactic ephedrine is desirable for spinal anaesthesia, especially in ASA III patients.

16. **RS Emmett, AM Cyna, M Andrew, SW Simmons et al**[47]did a cochrane review on techniques for preventing hypotension during spinal anaesthesia for caesarean section and concluded that no intervention reliably prevents hypotension during spinal anaesthesia for caesarean section. No conclusions are drawn regarding rare adverse effects of interventions due to their probable low incidence and the small numbers of women studied. Further trials are recommended, in particular assessing a combination of the beneficial interventions, ie colloid or crystalloid preloading, ephedrine administration and leg compression with bandages, stockings or inflatable boots.

17. **Coe AJ, Revanäs B. Et al**[48] did a study ;Is crystalloid preloading useful in spinal anaesthesia in the elderly? And concluded that Crystalloid preloading had no effect on the incidence of hypotension after spinal anaesthesia in fit, elderly patients.

18. ***Engberg G, Wiklund L.*****et al**[49] Did a study on the use of subcutaneous ephedrine premedication for prevention of arterial hypotension during epidural blockade and concluded that ephedrine premeditation prevented marked hypotension following epidural blockade in all groups, the pressor effect being most pronounccd when adrenaline was included in the local anaesthetic solution.

19. **W. S. Chan et al**[50] did a study on prevention of hypotension during spinal anaesthesia for caesarean section: ephedrine infusion versus fluid preload and concluded that prophylactic ephedrine infusion alone is at least as good as fluid preload alone in combating the hypotension associated with spinal anaesthesia for caesarean section.

20. **R.JACKSON et al**[51] did a comparison study on volume pre loading and prophylactic ephedrine infusion and concluded that volume preloading is not essential to prevent spinal induced hypotension in caesarean section.

AIM OF THE STUDY

To study the effects of various doses of dopamine as continuous intravenous infusion on the intraoperative hemodynamics in patients undergoing elective surgeries under spinal anaesthesia and to decide about the appropriate dose of dopamine needed to prevent hypotension in patients undergoing surgeries under spinal anaesthesia.

PRIMARY OBJECTIVE

To compare the various parameters observed during infusion of various doses of dopamine, like

- Heart rate
- Systolic blood pressure
- Diastolic blood pressure
- Mean arterial pressure

SECONDARY OBJECTIVE

To compare the Urine output in all 4 groups.

MATERIALS AND METHODS

PATIENT SELECTION :

After getting approval from the Ethical committee of Govt.Kilpauk medical college and getting written informed consent from patients/relatives,120 patients of ASA I & II who underwent elective surgeries under spinal anaesthesia at Government Kilpauk Medical College Hospital and Government Royapettah Hospital, were enrolled in this study group.

INCLUSION CRITERIA

- Patients of age between 25 to 65years
- height >150cm and <170cm,
- weight 40-75kg
- Patients undergoing elective surgeries under spinal anaesthesia requiring sensory blockade level of T6 and below
- ASA I & ASA II
- Consent from patient.

EXCLUSION CRITERIA

- ASAIII & ASA IV
- Patients who are known allergic to study drugs

- Patients having contraindications for spinal anaesthesia
- Emergency surgeries
- Valvular heart disease
- Ischaemic heart disease
- Hypertension
- Metabolic disorders
- Anaemic patient
- Prolonged surgeries for more than 2 hours
- Surgeries requiring sensory blockade above T6
- Surgeries causing major fluid shifts/blood loss >10% of blood volume
- Surgeries requiring other than supine position
- severe liver or kidney insufficiency
- history of hyperthyroidism/hypothyroidism
- recent administration of tricyclic antidepressants or monoamine oxidase inhibitors
- History of type2 DM >5yrs
- Patients not willing to take part in study

GROUPS :

a. Group A :30 patients who do not receive dopamine infusion during surgery
b. Group B : 30 patients who receive continuous intravenous Dopamine infusion @3mcg/kg/minute
c. Group C : 30 patients who receive continuous intravenous Dopamine infusion @5mcg/kg/minute
d. Group D : 30 patients who receive continuous intravenous Dopamine infusion @7mcg/kg/minute

METHODOLOGY :

This study was designed as a randomized control study.Patients were preoperatively evaluated,clinically examined and proper investigations were done prior to assessment. Procedure was explained in detail and written consent was obtained. After ascertaining the inclusion criteria,preoperative investigations were recorded.

Anesthesia procedure

The procedure was carried out in the theatre where facilities for resuscitation were available. All patients were deprived of food for overnight before surgery.Patients were randomly selected and divided into Group A, GroupB, Group C and Group D to receive intravenous dopamine infusion @ 0, 3, 5, and 7 mcg/kg/minute respectively. Intravenous cannulation was carried out with 18G venflon.All patients in four groups received inj.midazolam 2mg and inj.ondansetron 4mg intramuscularly half an hour prior to shifting to operating room.All the patients were preloaded with 15ml/kg of ringerlactate solution half an hour prior to subjecting to spinal anaesthesia.Vitals such as BP, Pulse, Respiratory rate, SPO2 were measured before the patient is being shifted to operating room. After shifting to operating room all the patients were subjected to spinal anaesthesia in lateral decubitus position, under sterile aseptic precautions using 25G QUINKE'S needle at L3-L4 space and 3.5ml of 0.5% bupivacaine[heavy] was given.

Intraoperatively group A, B,Cand D patients received dopamine infusion @0,3,5,7 mcg/kg/minute respectively soon after positioning the

patient. Level of sensory blockade checked at 5 minutes after sub arachnoid block.All the 4 group patients received crystalloid infusion intraoperatively @ 10ml/kg/hr.If any patients in group A,B,C,D had Hypotension (defined as a decrease in systolic arterial pressure (SAP) more than 20% below baseline and to below 100 mm Hg) intraoperatively,they received incremental doses of ephedrine(6mg) iv bolus as required.

Anesthesia monitoring

Pulse rate, blood pressure,mean arterial pressure, ECG, SpO2 were recorded at varying intervals[T1(1^{st} minute),T2(2^{nd} minute),T3,T4,T5]then every 5 minutes upto 30 minutes, then every 10 minutes till the end of the surgery. Urine output was measured every 60 minutes.Then the data were collected and analysed.

The parameters studied are

1. Systolic pressure
2. Diastolic pressure
3. Mean arterial pressure
4. heart rate
5. Urine output

Monitors used

- Pulse oximetry
- NIBP(Arterial blood pressure was measured by oscillometric method)
- ECG(3 LEAD ECG)

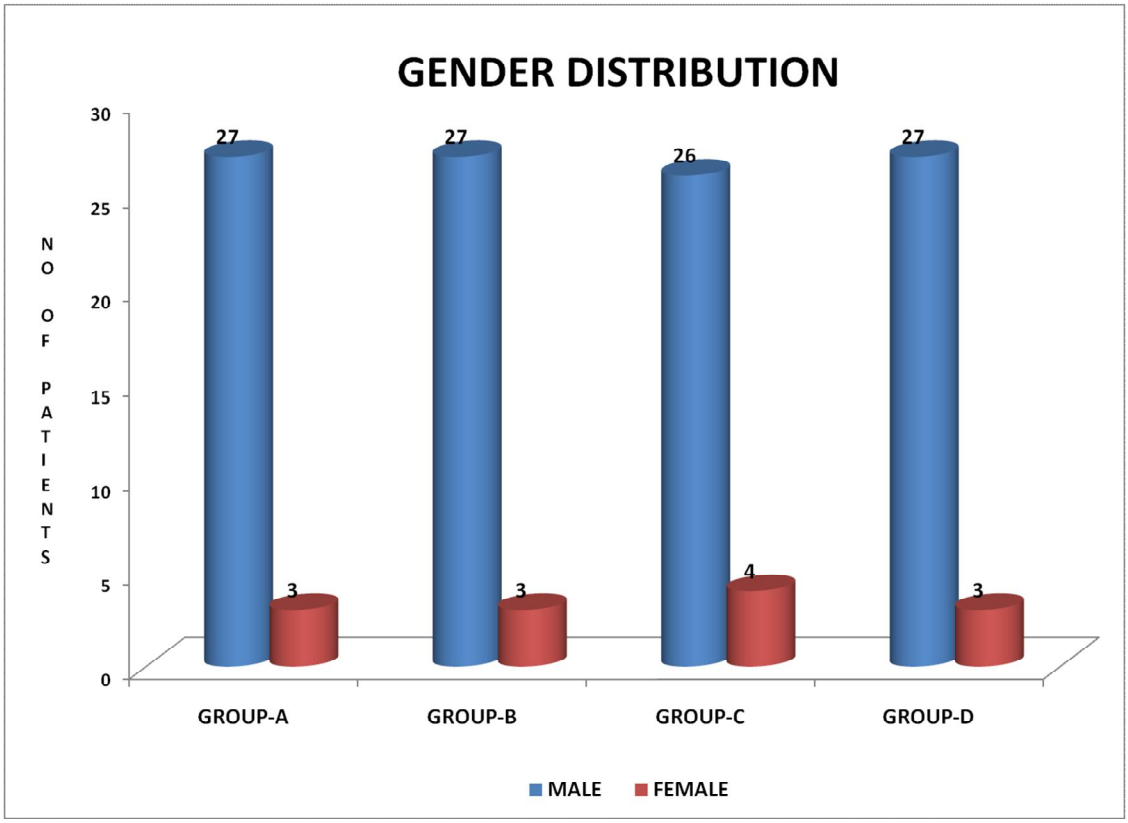
Data analysis

Statistical analyses were performed using SPSS 23.0 (SPSS Inc., IBM,Chicago, IL, USA). Sample size estimation was determined by power analysis with one-way analysis of variance (ANOVA).sample size of 120 subjects (n=30 per group) was determined to be sufficient to detect differences in the means among the 4 groups, with a statistical power greater than 90%. All numerical values are presented as the mean and standard deviation (SD).

Repeated-measures ANOVA was used to compare differences within the same group. One-way ANOVA was used to compare differences among groups. Categorical data were compared by the chi-squared test. Differences with $p<0.05$ were considered to be statistically significant.

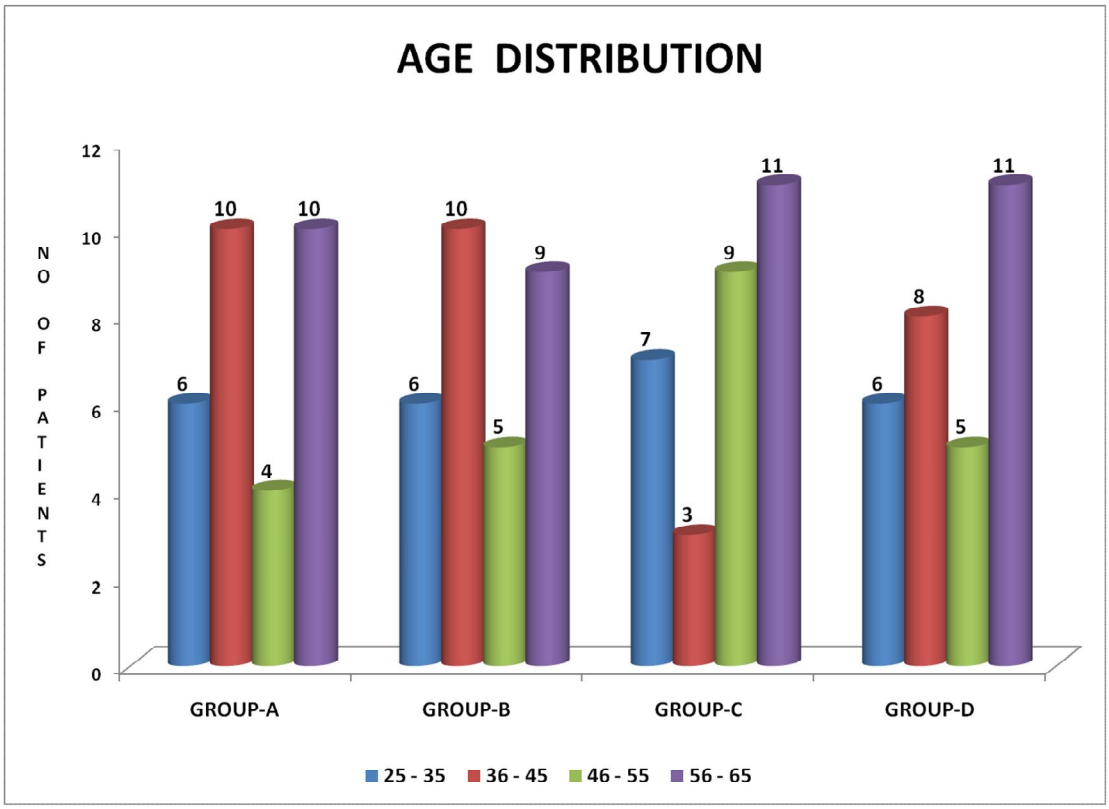
OBSERVATION AND RESULTS

- A total of 120 patients of ASA- PS1/PS2 were studied in this study.
- Thirty patients were enrolled into each of the four groups (A,B,C and D).
- There was no statistical significance between the four groups when the demographic parameters like age distribution, sex distribution,weight and height of the patients and ASA status classification were compared.
- The comparison of parameters like, pre operative systolic blood pressure, diastolic blood pressure , mean arterial pressure,and heart rate was also found to be statistically insignificant between the four groups.
- Sensory blockade level was also comparable between all groups and statistically insignificant.
- All the 120 patients underwent similar procedures which lasted for less than 120 minutes with insignificant blood loss.



GENDER DISTRIBUTION

Gender	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
MALE	3	10.00	3	10.00	4	13.33	3	10.00
FEMALE	27	90.00	27	90.00	26	86.67	27	90.00
TOTAL	30	100	30	100	30	100	30	100

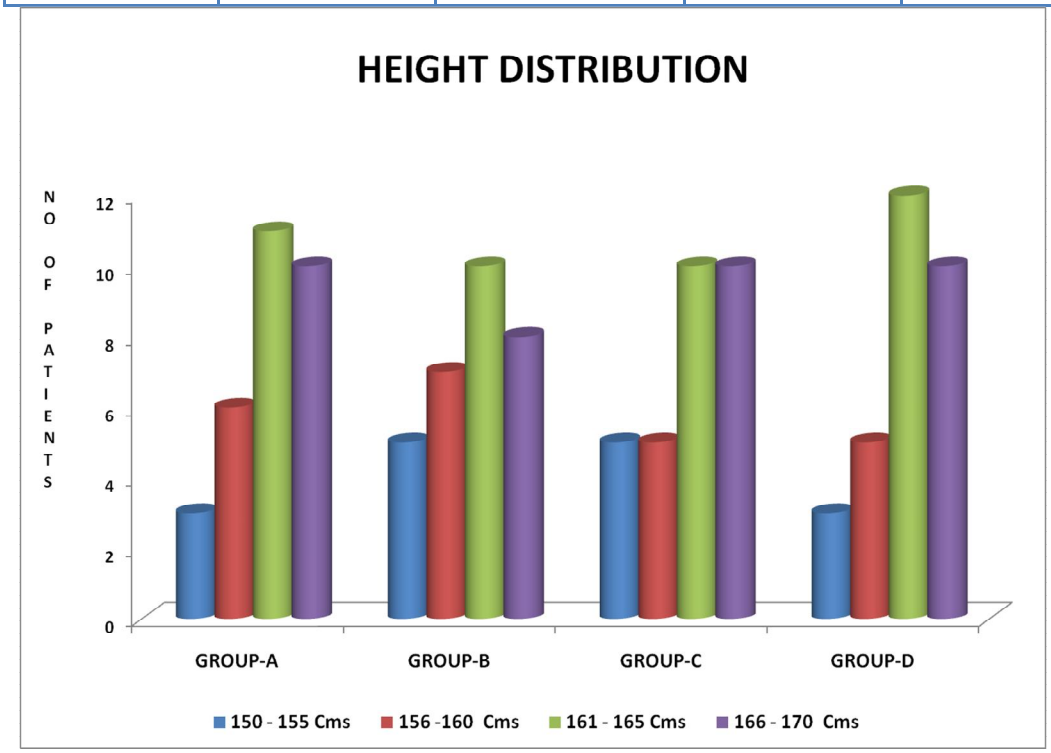


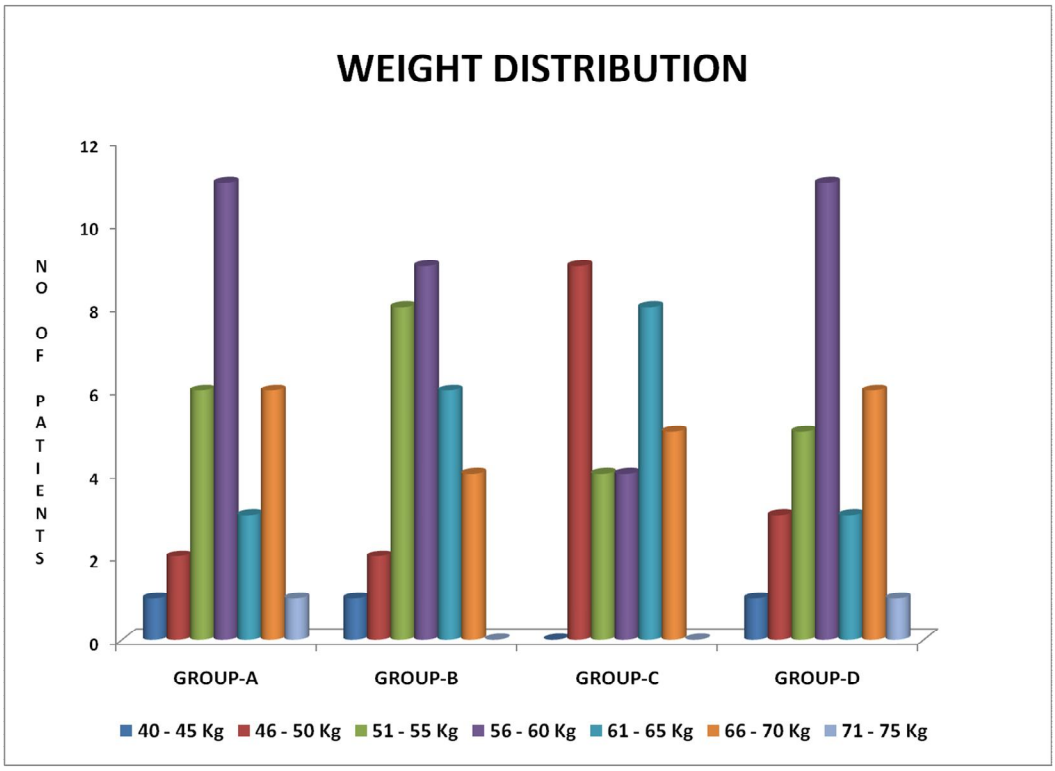
AGE GROUP

Age Group	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
25 - 35	6	20.00	6	20.00	7	23.33	6	20.00
36 - 45	10	33.33	10	33.33	3	10.00	8	26.66
46 - 55	4	13.34	5	16.67	9	30.00	5	16.67
56 - 65	10	33.33	9	30.00	11	36.67	11	36.67
TOTAL	30	100	30	100	30	100	30	100
Mean		47.60		46.77		48.10		47.83
SD		13.19		12.91		14.11		13.27

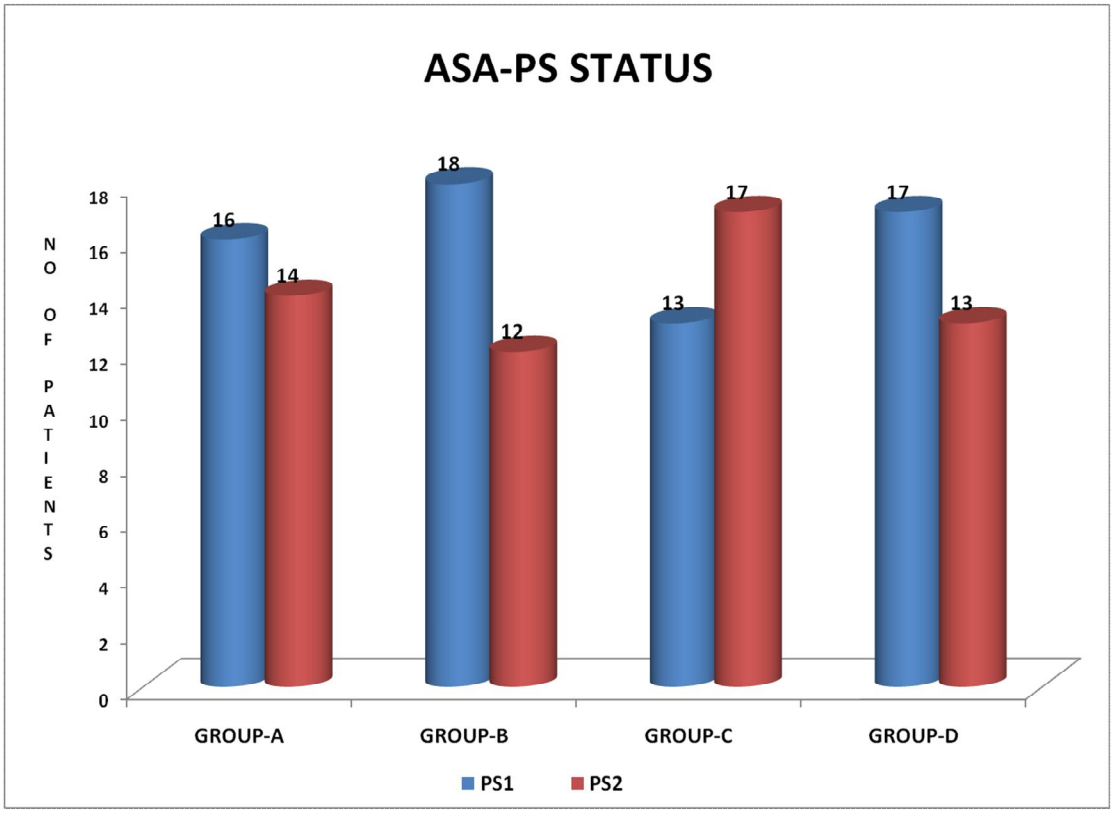
Height Distribution

Height (in Cms)	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
150 - 155	3	10.00	5	16.67	5	16.67	3	10.00
156 -160	6	20.00	7	23.33	5	16.67	5	16.67
161 - 165	11	36.67	10	33.33	10	33.33	12	40.00
166 - 170	10	33.33	8	26.67	10	33.33	10	33.33
TOTAL	30	100	30	100	30	100	30	100
Mean	163.23		161.57		163.13		163.27	
SD	5.22		5.39		5.31		5.05	
T-value Group A vs			1.22		0.07		0.03	
p-value			0.23		0.94		0.98	





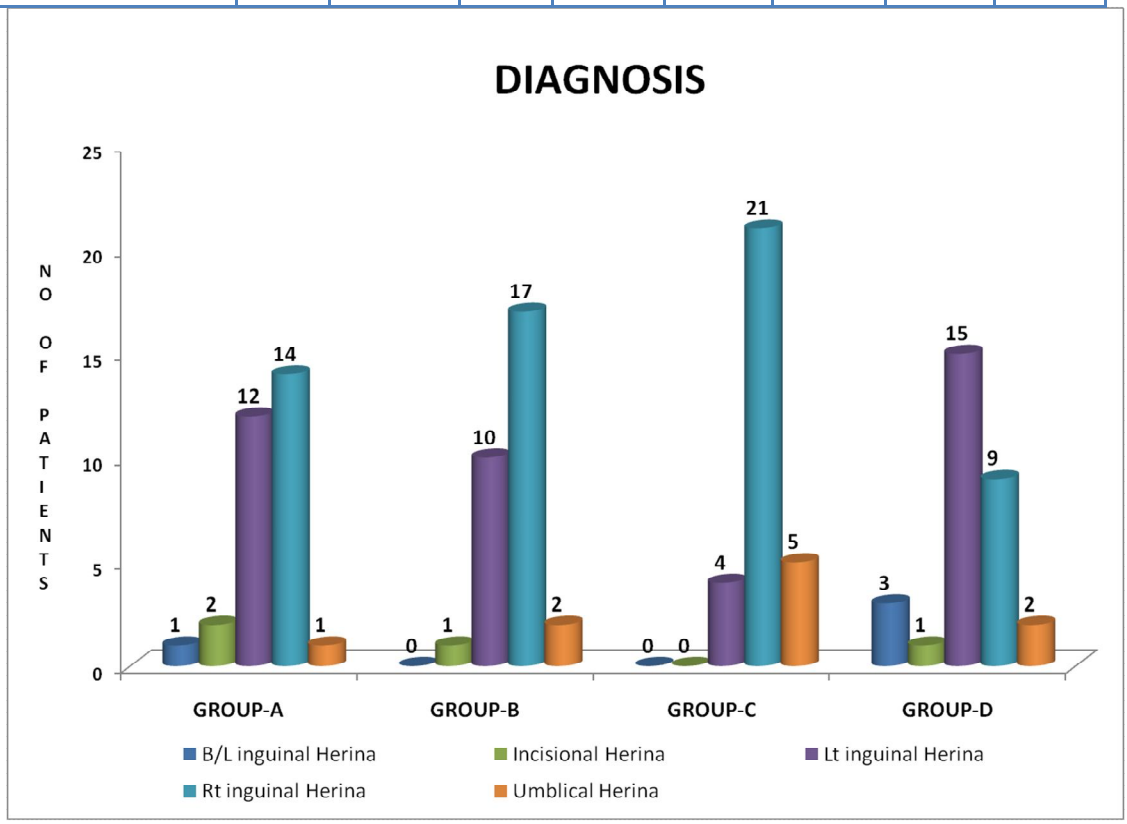
Weight in Kg	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
40 - 45	1	3.33	1	3.33	0	0.00	1	3.33
46 - 50	2	6.67	2	6.67	9	30.00	3	10.00
51 - 55	6	20.00	8	26.67	4	13.33	5	16.67
56 - 60	11	36.67	9	30.00	4	13.33	11	36.67
61 - 65	3	10.00	6	20.00	8	26.67	3	10.00
66 - 70	6	20.00	4	13.33	5	16.67	6	20.00
71 - 75	1	3.33	0	0.00	0	0.00	1	3.33
TOTAL	30	100	30	100	30	100	30	100
Mean		59.30		58.20		58.33		59.57
SD		6.95		6.01		6.87		7.26
t-value Group A vs				0.66		0.54		0.15
p-value				0.52		0.59		0.89



ASA-PS STATUS

ASA	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
PS1	16	53.33	18	60.00	13	43.33	17	56.67
PS2	14	46.67	12	40.00	17	56.67	13	43.33
TOTAL	30	100	30	100	30	100	30	100

Diagnosis	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
B/L inguinal Hernia	1	3.33	0	0	0	0	3	10.00
Incisional Herina	2	6.66	1	3.33	0	0	1	3.33
Lt inguinal Herina	12	40.00	10	33.33	4	13.33	15	50.00
Rt inguinal Herina	14	46.67	17	56.67	21	70.00	9	30.00
Umblical Herina	1	3.33	2	6.67	5	16.67	2	6.67
TOTAL	30	100	30	100	30	100	30	100



Systolic Blood Pressure

	GROUP-A	GROUP-B	GROUP-C	GROUP-D	Significance (p value)		
	Mean± SD	Mean± SD	Mean± SD	Mean± SD	A vs B	A vs C	A vs D
Preop	126.27±13.92	128.30±06.94	125.10±11.03	126.13±11.41	*0.48	*0.72	*0.96
T1	126.90±15.01	122.33±05.90	127.27±10.68	116.57±16.48	*0.13	*0.91	0.01
T2	114.60±19.82	114.50±10.47	129.17±11.56	114.00±17.17	*0.98	0.0001	*0.90
T3	109.57±18.81	111.47±12.36	133.33±15.43	119.87±19.56	*0.65	0.0001	0.04
T4	109.57±18.81	111.47±12.36	133.33±15.43	119.87±19.56	*0.62	0.0001	0.02
T5	108.20±15.12	105.20±14.92	128.77±12.76	123.20±23.15	*0.44	0.0001	0.004
T10	109.73±15.59	103.90±16.12	127.30±13.61	124.77±20.84	*0.16	0.0001	0.002
T15	108.67±14.26	109.03±11.60	123.67±12.06	124.60±19.91	*0.91	0.0001	0.001
T20	109.70±12.69	110.27±12.86	124.87±11.11	126.70±22.47	*0.86	0.0001	0.001
T25	107.80±13.79	109.70±12.52	125.37±14.28	136.50±25.97	*0.58	0.0001	0.0001
T30	108.97±13.83	112.87±11.46	121.43±14.09	133.23±21.34	*0.24	0.001	0.0001
T40	108.13±14.35	114.70±11.11	124.47±12.13	128.23±15.01	0.05	0.0001	0.0001
T50	110.90±14.31	115.40±10.10	126.90±13.17	127.80±13.69	*0.17	0.0001	0.0001

T60	112.40±14.24	114.23±9.22	127.33±12.08	125.73±6.57	*0.56	0.0001	0.0001
T70	105.26±22.70	117.90±11.91	124.93±14.51	120.50±5.13	0.01	0.0001	0.001
T80	112.80±13.03	119.73±9.04	124.77±13.72	123.10±3.48	0.02	0.002	0.0001
T90	111.05±12.52	119.47±8.36	124.96±9.09	120.13±2.16	0.01	0.0001	0.0001
T100	115.53±13.06	121.37±4.44	122.29±8.31	119.63±4.86	*0.08	*0.08	*0.13

*** Not Significant**

- When systolic blood pressure was compared between group A and the other 3 groups,there was significant statistical difference between group A and C, A and D,from T2 to T90 minutes.

Diastolic Blood Pressure

	GROUP-A	GROUP-B	GROUP-C	GROUP-D	Significance		
	Mean± SD	Mean± SD	Mean± SD	Mean± SD	A vs B	A vs C	A vs D
Pre op	84.63± 9.07	82.53± 2.43	83.07± 4.46	81.76± 8.40	*0.23	*0.40	*0.21
T1	79.70± 9.52	78.13± 2.89	81.13±10. 58	74.43±16. 58	*0.39	*0.58	*0.14
T2	72.37±12. 45	72.23± 7.20	77.83± 9.25	71.33±16. 85	*0.96	*0.06	*0.79
T3	69.43±10. 48	68.67± 8.97	79.97± 9.74	74.93±14. 76	*0.76	0.000 1	*0.10
T4	70.67±11. 80	67.50±10. 20	76.23± 9.80	75.83±14. 77	*0.27	0.05	*0.14
T5	69.47±10. 49	65.20±12. 66	74.60±10. 99	69.73±16. 27	*0.16	*0.07	*0.94
T10	69.00±10. 69	65.57±11. 06	74.53± 9.73	71.27±14. 55	*0.23	0.04	*0.49
T15	68.33±11. 45	70.63±12. 61	72.87± 8.59	71.47±15. 04	*0.46	*0.09	*0.37
T20	69.73±10. 73	69.23±10. 26	73.90± 8.16	71.90±14. 61	*0.85	*0.10	*0.52
T25	68.03±10. 80	67.57± 6.68	73.57±11. 02	73.40±14. 33	*0.84	0.05	*0.11
T30	69.00±10. 58	68.00± 7.46	71.97±11. 20	73.67±14. 50	*0.67	*0.30	*0.16
T40	67.37±10. 36	70.17± 7.23	71.60±11. 34	70.83± 9.82	*0.23	*0.14	*0.19
T50	69.30±11. 38	71.83± 6.81	72.17± 8.65	70.30±11. 52	*0.30	*0.28	*0.74
T60	71.57±11. 29	71.23±7.2 0	73.03± 9.21	74.20±10. 64	*0.89	*0.58	*0.36
T70	73.37±11.	69.03±11.	70.20±	71.10±	*0.15	*0.22	*0.37

	10	26	7.99	7.78			
T80	71.84±10.69	72.53±6.24	76.37±6.45	73.73±8.09	*0.77	*0.06	*0.46
T90	70.95±9.61	73.93±4.79	75.35±4.69	70.90±6.74	*0.15	0.05	*0.98
T100	72.94±10.67	78.79±7.74	78.35±4.17	73.43±4.69	*0.07	*0.06	*0.83

*** Not Significant**

- When diastolic blood pressure of the control group was compared with the other three groups B,C and D there was no significant difference statistically.

Mean arterial Pressure

	GROUP-A	GROUP-B	GROUP-C	GROUP-D	Significant		
	Mean± SD	Mean± SD	Mean± SD	Mean± SD	A vs B	A vs C	A vs D
Pre op	102.33±9.22	101.40±5.65	101.27±8.46	102.37±8.07	*0.64	*0.64	*0.99
T1	94.50±9.08	92.13±4.09	94.90±9.44	86.70±16.17	*0.20	*0.87	0.03
T2	85.60±14.02	84.80±8.43	93.67±7.50	88.43±15.93	*0.79	0.01	*0.47
T3	82.67±12.51	82.13±10.53	96.67±8.25	91.40±16.26	*0.86	0.0001	0.02
T4	82.13±12.88	81.17±11.29	95.87±7.32	89.20±16.61	*0.76	0.0001	*0.07
T5	81.03±11.28	77.47±14.02	93.80±7.00	85.87±18.87	*0.28	0.0001	*0.23
T10	81.03±10.16	77.23±13.71	93.63±9.26	88.27±16.11	*0.23	0.0001	0.04
T15	80.87±10.47	83.87±14.53	89.90±8.13	89.70±17.89	*0.36	0.0001	0.02
T20	80.63±10.16	82.50±11.22	89.07±7.83	91.20±21.30	*0.50	0.001	0.02
T25	79.83±10.78	81.27±8.84	92.13±10.05	94.70±19.18	*0.58	0.0001	0.0001
T30	80.80±10.12	82.27±8.67	87.80±8.70	94.80±17.81	*0.55	0.01	0.0001
T40	80.00±10.54	83.97±8.43	89.43±7.33	86.93±13.78	*0.11	0.0001	0.03
T50	81.63±10.79	84.70±7.63	90.23±7.20	88.40±13.35	*0.21	0.001	*0.34
T60	84.20±10.44	84.10±7.94	92.30±6.95	88.23±8.86	*0.97	0.001	*0.11
T70	80.89±12.	84.57±10.	88.23±	85.17±	*0.24	0.01	*0.11

	86	57	6.69	5.84			
T80	84.60± 9.86	85.77± 8.10	91.77± 7.65	88.57± 5.71	*0.63	0.01	*0.07
T90	85.10±10. 39	86.90± 7.14	94.46± 7.81	85.10± 1.42	*0.47	0.001	*1.00
T100	87.59±11. 53	90.68± 8.08	95.53± 8.66	86.47± 1.14	*0.35	0.03	*0.60

*** Not Significant**

- When mean arterial pressure of the three groups B,C and D,compared with control group A,there was statistically significant difference seen between the groups A and C(T2-T100),A and D(T3,T10-T40).

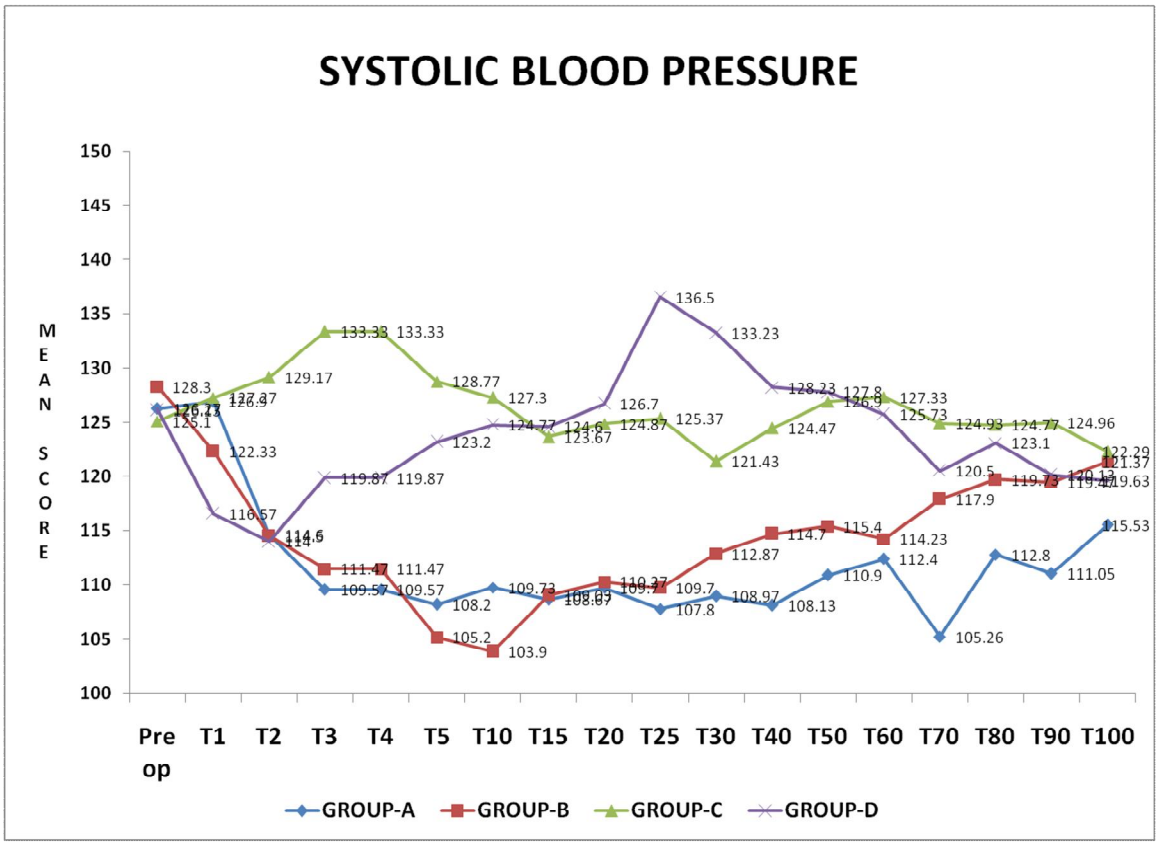
Heart Rate

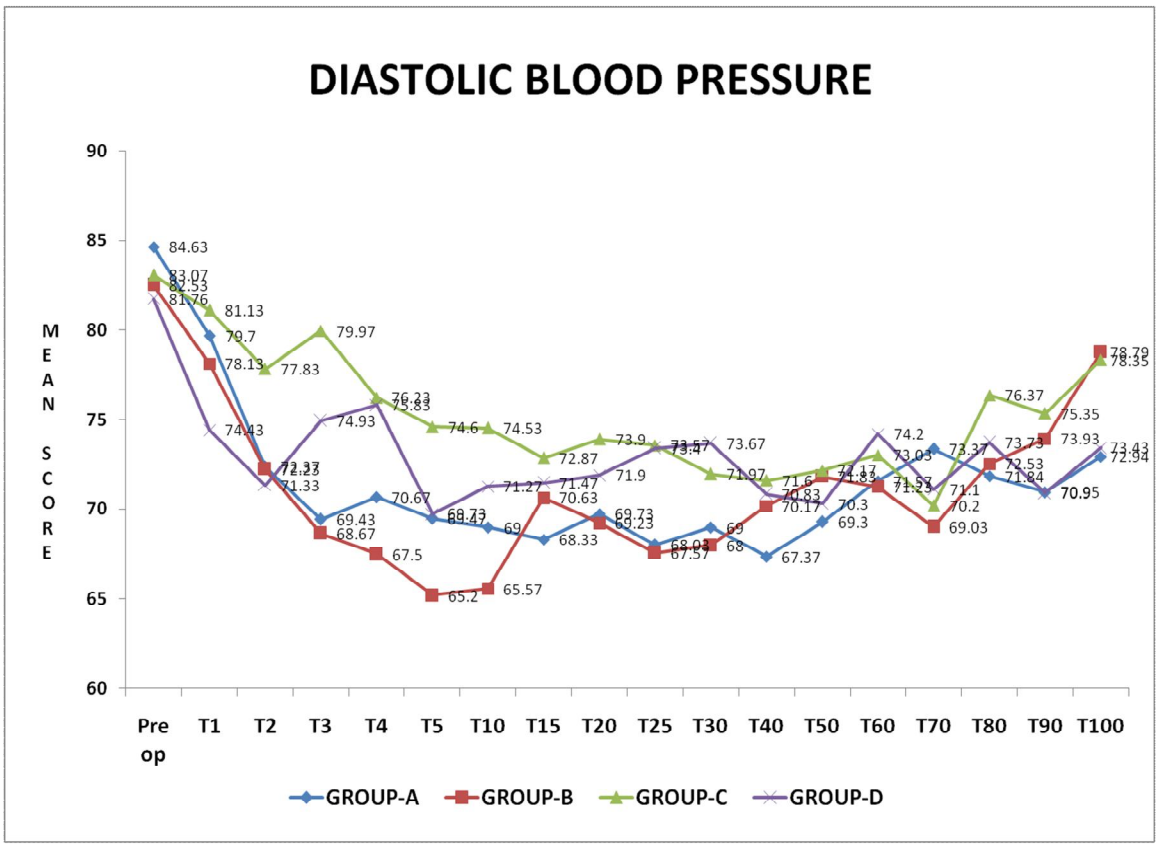
	GROUP-A	GROUP-B	GROUP-C	GROUP-D	Significant		
	Mean± SD	Mean± SD	Mean± SD	Mean± SD	A vs B	A vs C	A vs D
Pre op	84.37±12.36	83.43±10.01	84.47± 8.22	80.43±11.39	*0.74	*0.97	*0.20
T1	83.77±14.21	88.47± 9.02	86.03± 8.77	79.57±14.04	*0.13	*0.46	*0.25
T2	81.67±15.89	88.20± 6.55	86.10± 8.22	72.93± 8.66	0.04	*0.18	0.01
T3	80.53±15.96	80.60± 6.44	84.43±10.99	74.43± 8.42	*0.06	*0.28	*0.07
T4	78.73±16.05	90.87± 7.79	83.83±13.22	75.60±10.01	0.0001	*0.18	*0.37
T5	75.33±14.43	87.33± 8.78	80.43± 8.92	73.13± 8.63	0.0001	*0.11	*0.48
T10	73.10±13.62	86.47±10.52	79.80±12.91	72.33±10.23	0.0001	*0.06	*0.81
T15	71.67±12.63	80.50±10.33	78.63± 9.55	70.37±11.40	0.004	0.02	*0.68
T20	70.33±12.23	75.00±12.87	74.43±12.41	70.83± 9.65	*0.16	*0.20	*0.86
T25	68.40±10.44	74.20±13.28	72.13± 7.53	70.17± 8.89	*0.07	*0.12	*0.48
T30	67.83± 9.82	74.23±14.11	74.27± 9.17	73.27± 8.54	0.05	0.01	0.03
T40	66.20± 9.56	76.73±15.16	75.33± 7.99	70.53± 8.51	0.002	0.0001	*0.07
T50	67.27± 9.63	74.17±12.40	73.00± 7.41	70.73± 9.90	0.02	0.01	*0.18
T60	67.53±10.21	74.77±13.41	73.70± 8.00	74.60± 8.91	0.02	0.01	0.01
T70	67.33±18.	77.00±13.	72.57±	72.93±	0.03	*0.15	*0.16

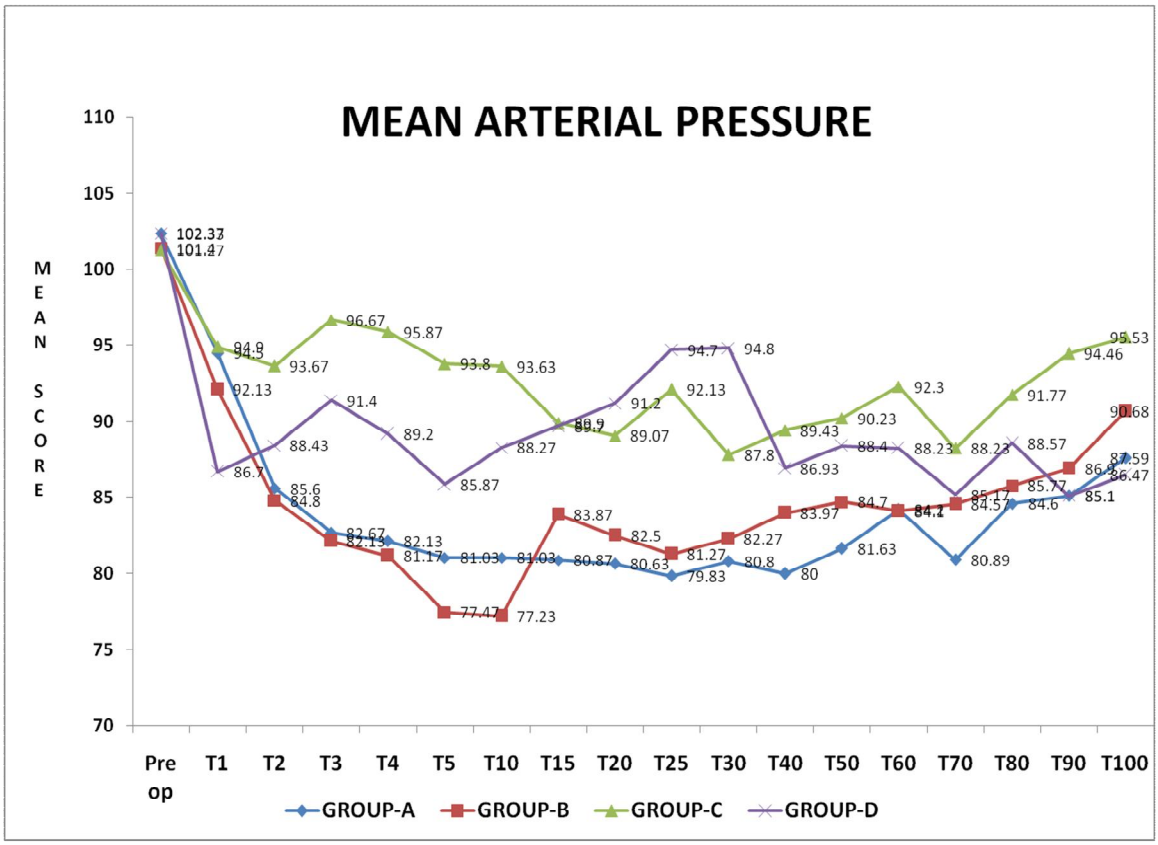
	82	29	4.97	9.98			
T80	67.20±10.34	76.87±14.18	76.60±5.64	73.10±11.74	0.01	0.0001	*0.06
T90	65.80±10.21	76.07±11.06	76.08±5.54	72.70±2.93	0.002	0.0001	0.001
T100	67.24±10.42	74.79±15.24	76.41±6.51	79.17±9.25	*0.10	0.004	0.0001

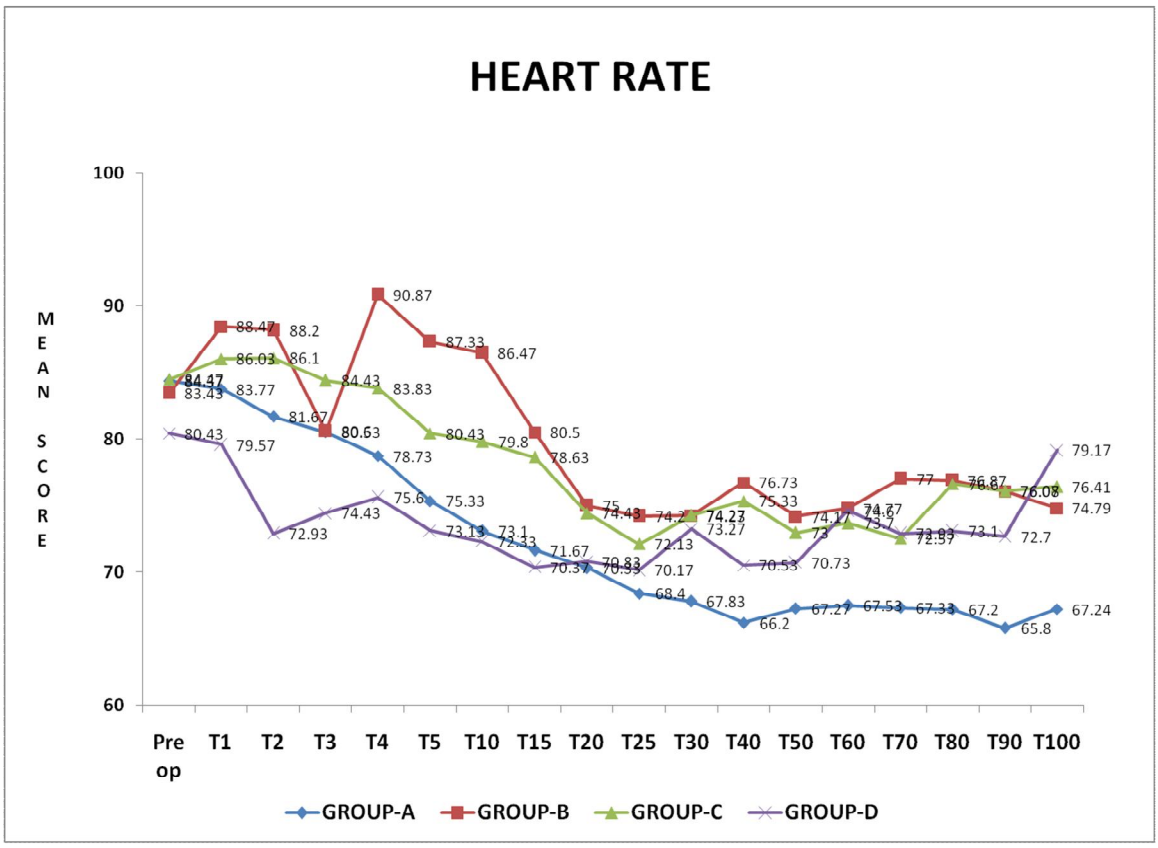
*** Not Significant**

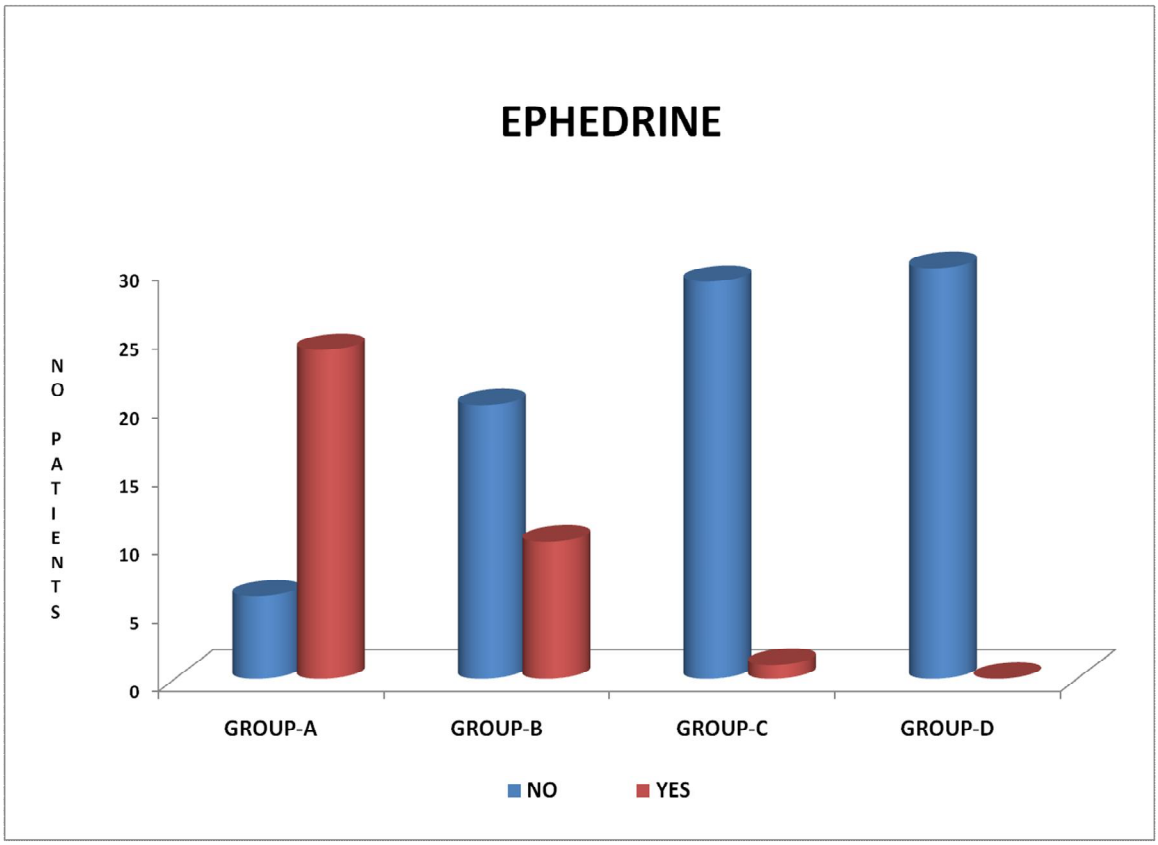
- When the heart rate of group A is compared with other three groups there was no consistent statistical difference till T30 in group A Vs C, A Vs D.But after T30 minutes there was significant fall in the heart rate in group A when compared to other 3 groups.


SYSTOLIC BLOOD PRESSURE
MEAN SCORE
150
145
140
135
130
125
120
115
110
105
100
Pre op
T1
T2
T3
T4
T5
T10
T15
T20
T25
T30
T40
T50
T60
T70
T80
T90
T100
GROUP-A
GROUP-B
GROUP-C
GROUP-D


DIASTOLIC BLOOD PRESSURE
MEAN SCORE
90
85
80
75
70
65
60
Pre op
T1
T2
T3
T4
T5
T10
T15
T20
T25
T30
T40
T50
T60
T70
T80
T90
T100
GROUP-A
GROUP-B
GROUP-C
GROUP-D

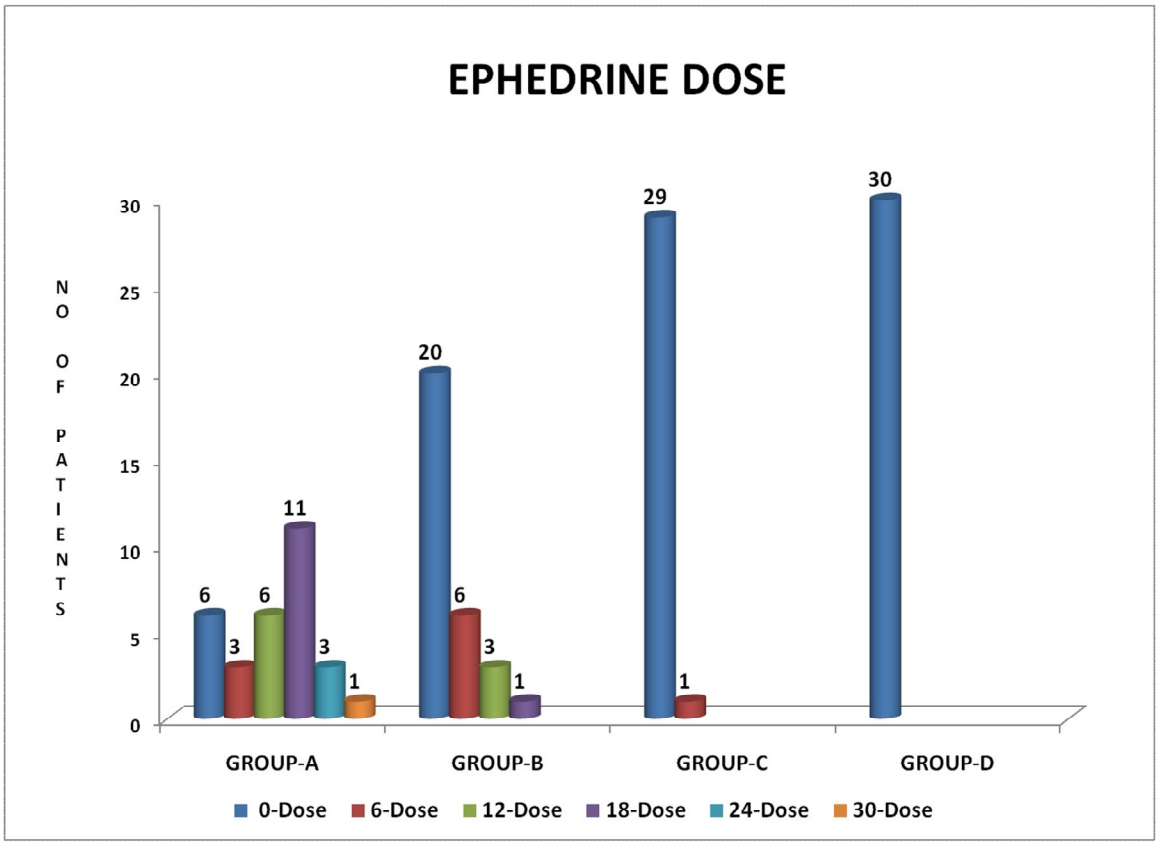

MEAN ARTERIAL PRESSURE
MEAN SCORE
110
105
100
95
90
85
80
75
70
Pre op
T1
T2
T3
T4
T5
T10
T15
T20
T25
T30
T40
T50
T60
T70
T80
T90
T100
GROUP-A
GROUP-B
GROUP-C
GROUP-D


HEART RATE
MEAN SCORE
100
90
80
70
60
Pre op
T1
T2
T3
T4
T5
T10
T15
T20
T25
T30
T40
T50
T60
T70
T80
T90
T100
GROUP-A
GROUP-B
GROUP-C
GROUP-D



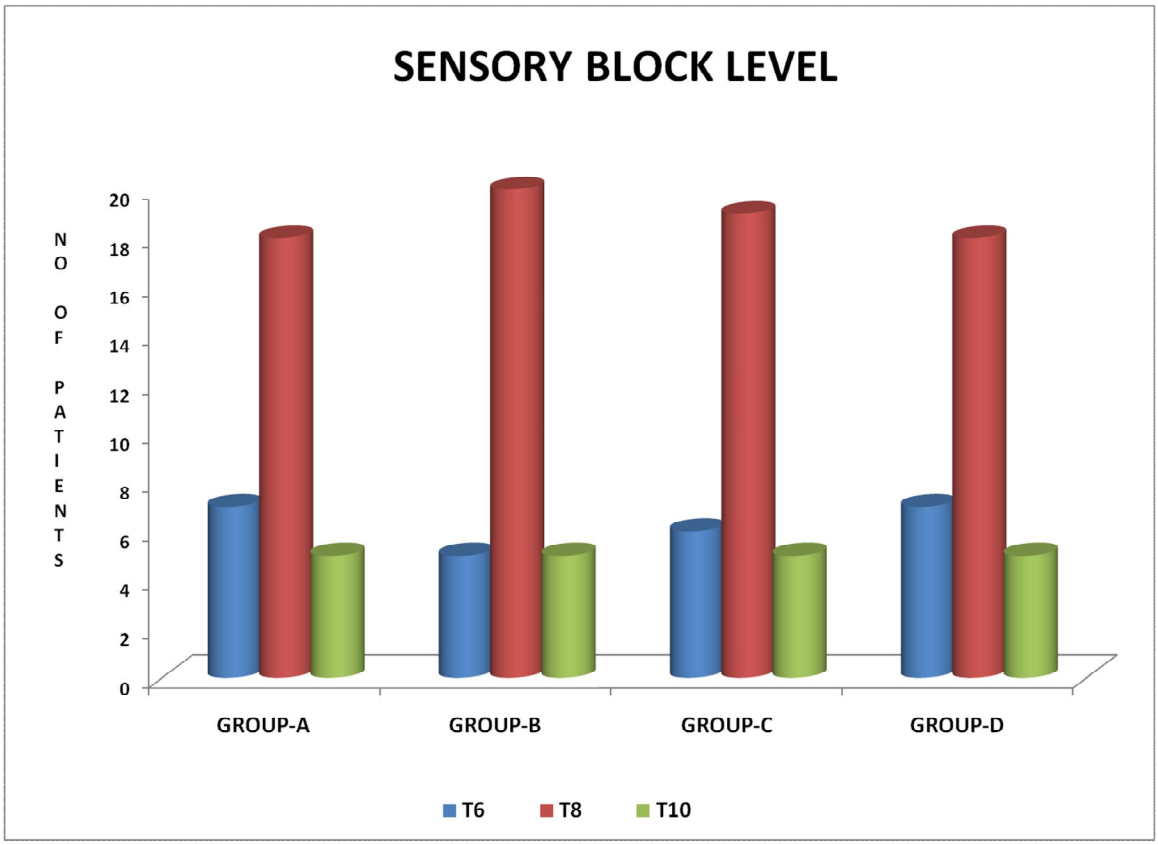
Ephedrine	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
NO	6	20.00	20	66.66	29	96.66	30	100
YES	24	80.00	10	33.33	1	3.33	0	0
TOTAL	30	100	30	100	30	100	30	100

- In group A 24 patients required ephedrine intravenous boluses.
- In group B 10 patients required ephedrine intravenous boluses.
- In group C 1 patient and in group D none required ephedrine respectively.



EPHEDRINE DOSE

Ephedrine Dose in mg	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
0	6	20.0	20	66.66	29	96.66	30	100
6	3	10.00	6	20.0	1	3.33	-	-
12	6	20.00	3	10.00	-	-	-	-
18	11	36.67	1	3.33	-	-	-	-
24	3	10.00	-	-	-	-	-	-
30	1	3.33	-	-	-	-	-	-
TOTAL	30	100	30	100	30	100	30	100



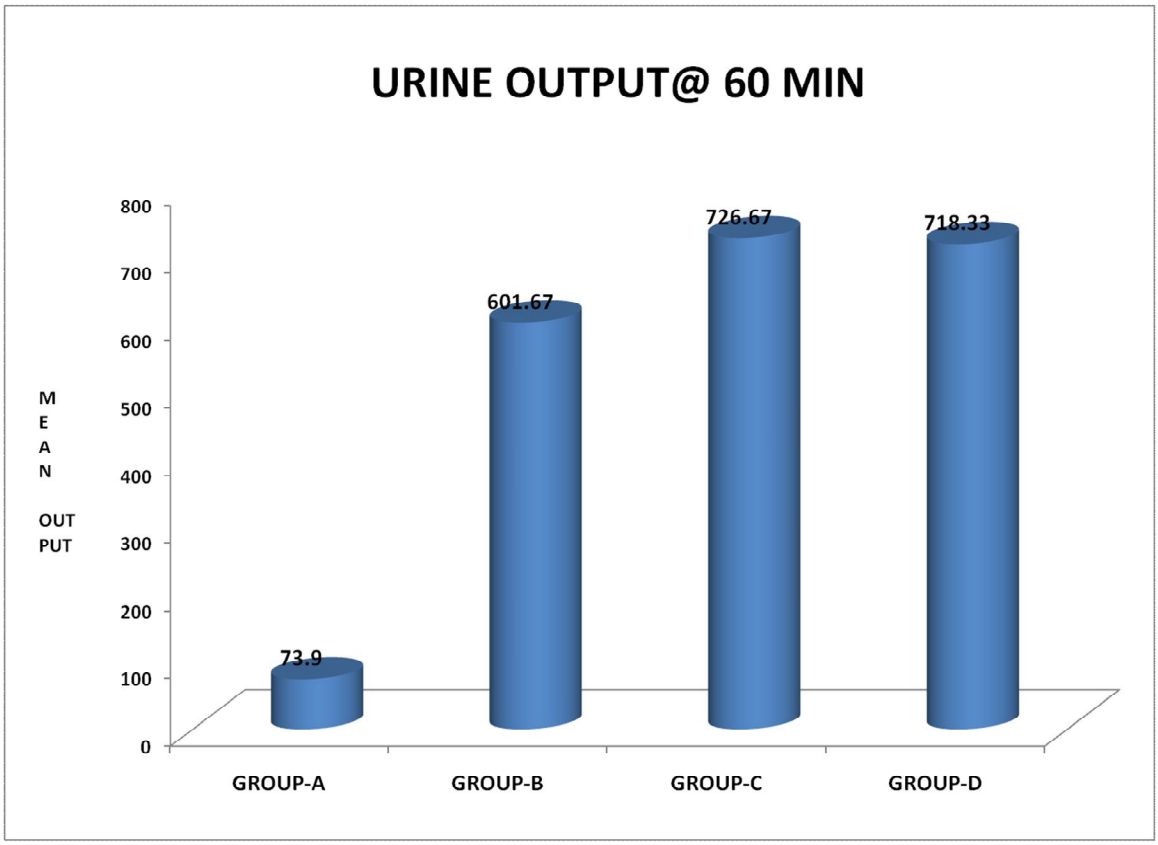
Sensory block level

	GROUP-A		GROUP-B		GROUP-C		GROUP-D	
	N	%	N	%	N	%	N	%
T6	7	23.33	5	16.66	6	20.0	7	23.3
T8	18	60.00	20	66.67	19	63.33	18	60.00
T10	5	16.67	5	16.67	5	16.67	5	16.67
TOTAL	30	100	30	100	30	100	30	100

- The sensory blockade level was comparable between the four groups and it is statistically insignificant.

Group	Hypertension	Hypotension	Bradycardia Hr<50/mt	Desaturation Spo2<95	Tachycardia Hr >100/mt
A	0/30	24/30	0	0	0
B	0/30	10/30	0	0	0
C	0/30	1/30	0	0	0
D	14/30	0/30	0	0	0

- In group A 24 patients, in group B 10 patients, in group C 1 patient and in group D none had hypotension respectively.
- In group D 14 patients had hypertension which needed tapering down of dopamine infusion to 5mcg/kg/min.
- None had bradycardia or tachycardia, but group A patients had a statistically significant lower heart rate after T30, when compared to group B, C and D.



- There was a significant difference in urine output noted between the control group A and groups which received dopamine infusion.
- The average volume of urine in control group is 73.9 ml at the end of 60 minutes, but it was around 10 times more in group B , C and D.

DISCUSSION

With very few exceptions, the effects of spinal anesthesia on the cardiovascular system are almost entirely because of block of the preganglionic sympathetic fibers by the local anesthetic injected in the subarachnoid space .

Physiologic trespass is directly related to the intrathecal level of sympathetic denervation. The degree to which the spinal anesthetic alters the normal hemodynamic status, however, varies considerably.

Differences may be due to many factors, including the general state of health, age, intravascular fluid status, and concurrent medications. In general, more extensive sympathetic block produces more profound hemodynamic changes. The effects of sympathetic denervation are extensive, both on the arterial and venous vessels.

Hence sympathomimetics are the mainstay in the management of central neuraxial blockade induced hypotension.

In our study dopamine was used premptively as vasopressor in three different doses 3,5,7 mcg per kg per minute, to compare the intra operative hemodynamics of various doses of dopamine infusion.

Dopamine at a dose of 5mcg/kg/min increases the BP without obviously affecting the HR. Thus, it can be used to control

hemodynamics during spinal anesthesia, without increasing myocardial consumption.

We investigated the effects of continuous intravenous infusion of dopamine at 3, 5, and 7 µg/kg/min on the hemodynamic parameters of patients undergoing surgeries under spinal anesthesia. Systolic BP and MAP were higher in Groups C and D compared to Group A suggesting that continuous intravenous infusion of dopamine at 5 or 7 µg/kg/min is effective in maintaining hemodynamic stability during surgery.

HR was not increased in Group B,C or D when compared with Group A, suggesting that dopamine does not cause tachycardia even at a dose of 7mcg/kg/mt.

However, the hypertension incidence was higher in Group D than in any other group. Therefore, even though it maintains the hemodynamic stability, continuous intravenous infusion of 7 µg/kg/min dopamine may not be safe for patients.

Overall, our findings indicate that continuous intravenous infusion of 5µg/ kg/min dopamine may be safe and effective in controlling hemodynamic stability for patients undergoing surgeries under spinal anaesthesia.

In **Cabalum et al**[32] study Infusion of dopamine during the spinal hypotension corrected the disturbed circulatory parameters. Dopamine represented a useful agent in the management of spinal hypotension.

In our study also dopamine at a dose of 5mcg/kg/minute was able to maintain stable intraoperative systolic and mean arterial pressure when compared to ephedrine iv boluses .

The effects of dopamine on hemodynamic parameters were examined at several time points before and during surgery. In the control group, systolic BP,MAP was lower at T2–T100compared to T1, suggesting that spinal anesthesia induced hypotension in these patients were not well managed by intermittent ephedrine intravenous boluses.

Frazer RS Edwards GM. et al[43] **,Kang YG, Abouleish E, Caritis S. et al**[44]**,*Gajraj NM, Victory RA, Pace NA, Van Elstraete AC, Wallace DH*.et al**[45] **,*Hemmingsen C, Poulsen JA, Risbo A*. Et al**[46]. All the above four studies mentioned that prophylactic ephedrine administration prevented hypotension in patients underwent central neuraxial blockade but in our study control group received only iv intermittent boluses and they had a wide variation in systolic and mean arterial pressures when compared to dopamine infusion group C.

In Group B, systolic BP,MAP were low and not different from those in Group A, suggesting that continuous intravenous infusion of 3µg/kg/min dopamine did not effectively control hemodynamic stability.

Butterworth et al[34] studied the effectiveness of dopamine as possible alternative to ephedrine for the pharmacologic correction of the non cardiac circulatory sequelae of spinal anesthesia was demonstrated and **Lundberg et al**[35] found that myocardial contractility and arterial pressure were restored to pre-thoracic epidural analgesia values by dopamine at 5–10 $\mu g \cdot kg^{-1} \cdot min^{-1}$.

In our study also we found that dopamine infusion at a dose of 5mcg/kg/min can be used as a safe alternative to ephedrine iv boluses for spinal anaesthesia induced hypotension and dopamine infusion had a better cardiovascular stability when compared to ephedrine group and there was no tachycardia reported even at the dose of 7mcg/kg/min.

Coe AJ, Revanäs B. Et al[48] did a study using crystalloid preloading to assess usefulness in spinal anaesthesia in the elderly.And

concluded that Crystalloid preloading had no effect on the incidence of hypotension after spinal anaesthesia in fit, elderly patients.

In our study even after preloading with crystalloids@15ml/kg, group A patients had hypotension necessitating ephedrine intravenous boluses. But in group B, 10 patients required ephedrine bolus, in group C only one patient needed ephedrine bolus and in group D patients did not require ephedrine bolus.

Moreover patients in group B,C&D had an average urine output of >700 ml at the end of 60 minutes,indicating that patients received dopamine had a better hemodynamics compared to intermittent ephedrine boluses.

SUMMARY

In this study

- We observed that dopamine continuous infusion @ 5mcg/kg/min prevented spinal anaesthesia induced hypotension effectively without causing significant tachycardia.

- We observed that dopamine infusion @ 3mcg/kg/min was not able to maintain stable hemodynamics in patients undergoing surgeries under spinal anaesthesia.

- We observed that dopamine infusion @ 7mcg/kg/min was even though able to prevent spinal anaesthesia induced hypotension,it produced hypertension in significant number of patients.

- We observed that intermittent intravenous ephedrine boluses failed to prevent the spinal anaesthesia induced hypotension.

- We also observed that patients who received dopamine infusion had a higher urine output in all 3 doses when compared to control group.

CONCLUSION

With this study we conclude that preemptive dopamine continuous intravenous infusion at a dose of 5 mcg/kg/min effectively prevented spinal anaesthesia induced hypotension when compared to intravenous intermittent ephedrine boluses without any significant adverse effects.Hence with this study we recommend that dopamine @ 5mcg/kg/min infusion can be used as a safe alternative for intermittent ephedrine iv boluses.

BIBLIOGRAPHY

1. *Shimosato S, Etsten BE. The role of the venous system in cardiocirculatory dynamics during spinal and epidural anesthesia in man. Anesthesiology 1969; 30: 619-28.*

2. *Sancetto SM, Lynn RB, Simeone FA, Scott RW. Studies of the hemodynamic changes in humans following induction of low and high spinal anesthesia. General considerations of the problem. The changes in cardiac output, brachial arterial pressure, peripheral and pulmonary oxy- gen contents and peripheral blood flows induced by spinal anesthesia in humans not undergoing surgery. Circulation 1952; 6: 559-71.*

3. *Milwidsky H, de Vries A. Regulation of blood pressure during spinal anesthesia: observations on intramuscular pressure and skin temperature. Anesthesiology 1948; 9: 258-75.*

4. *Butterworth JF IV, Austin JC, Johnson MD, et al. Effect of total spinal anesthesia on arterial and venous responses to dopamine and dobutamine. Anesth Analg. 1987;66:209.*

5. *Cronenwett JL, Zelenock GB, Whitehouse WM Jr, et al. The effect of sympathetic innervation on canine muscle and skin blood flow. Arch Surg. 1983;118:420.*

6. *Otton PE, Wilson El.. The cardiocirculatory effects of upper thoracic epidural analgesia. Can Anaesth Soc J 1966; 13: 541-9.*

7. *McLean APH, Mulligan GW, Otton P, MacLean LD Hemodynamic alterations associated with epidural anesthe- sia: Surgery 1967; 62: 79-87.*

8. *Sancetta SM, Lynn RB, Simeone FA, et al. Circulation. 1952;6:559.*

9. *Pollard JB. Common mechanisms and strategies for prevention and treatment of cardiac arrest during epidural anesthesia. J Clin Anesth. 2002;14:52.*

10. *Eckenhoff JE, Hafkenschiel JH, Foltz EL, et al. Influence of hypotension on coronary blood flow, cardiac work, and cardiac efficiency. Am J Physiol. 1948;152:545.*

11. *Underwood SM, Glynn CJ. Sick sinus syndrome manifest after spinal anaesthesia. Anaesthesia. 1988;43:307.*

12. *Hackel DB, Sancetta SM, Kleinerman J. Effect of hypotension due to spinal anesthesia on coronary blood flow and myocardial metabolism in man. Circulation. 1956;13:92.*

13. *Klassen GA, Bramwell RS, Bromage PR, et al. Effect of acute sympathectomy by epidural anesthesia on the canine circulation. Anesthesiology. 1980;52:8.*

14. *Blomberg S, Emanuelsson H, Ricksten SE. Thoracic epidural anesthesia and central hemodynamics in patients with unstable angina pectoris. Anesth Analg. 1989;69:558.*

15. *Giasi RM, D'Agostino, Covino BG. Absorption of lidocaine following subarachnoid and epidural administration. Anesth Analg 1979; 58: 360–3.*

16. *Greene NM. Blood levels of local anesthetics during spinal and epidural anesthesia (Editorial). Anesth Analg 1979; 58: 357–9.*

17. *Harrison DC, Sprouse JH, Morrow AG. The antiarrhythmic properties of lidocaine and procaine amide: clinical and physiological studies of their cardiovascular effects in man. Circulation 1963; 28: 486–91.*

18. *Jorfeldt L, Löfström B, Pernow B, Wahren J, Widman B. The effect of local anaesthetics on the central circulation and respiration in man and dog. Acta Anaesthesiol Scand 1968; 12: 153–69.*

19. *Kleinerman J, Sancetta SM, Hackel DB. Effects of high spinal anesthesia on cerebral circulation and metabolism in man. J Clin Invest 1958; 37: 285–93.*

20. *Dohi S, Naito H, Takahashi T. Age-related changes in blood pressure and duration of motor block in spinal anesthesia. Anesthesiology 1979; 50: 319–23.*

21. *Kestin IG. Spinal anaesthesia in obstetrics. Br J Anaesth 1991; 66: 596-607.*

22. *Greene NM, Brull SJ. Physiology of Spinal Anesthesia, 4th ed. Baltimore: Williams & Wilkins. 1993: 85-199, 309-43, and 357-80*

23. *Baron J-F, Coriat R Mundler O, Fauchet M, Bousseau D, Viars P. Left ventricular global and regional function dur- ing lumbar epidural anesthesia in patients with and without angina pectoris. Influence of volume loading. Anesthesiol- ogy 1987; 66: 621-7.*

24. *Hahn RG. Blood volume at the onset of hypotension dur- ing TURP performed under epidural anaesthesia. Eur J Anaesthesiol 1993; 10: 219-25.*

25. *Hahn RG. Increased haemodilution in hypotension in- duced by epidural anaesthesia. Acta Anaesthesiol Scand 1993; 37: 357-60.*

26. *Wennberg E,Frid I, Haljamiie H, NorOn H. Colloid (3% dextran 70) with or without ephedrine infusion for cardio- vascular stability during extradural Caesarean section. Br J Anaesth 1992; 69: 13-8.*

27. *Zorko N, Kamenik M, Starc V. The effect of Trendelenburg position, lactated ringer's solution and hydroxyethyl starch solution on cardiac output after spinal anesthesia. Anesth Analg. 2009;108:655-9.*

28. *Shin BS, Kim CS, Sim WS, et al. A comparison of the effects of preanesthetic administration of crystalloid versus colloid on intrathecal spread of isobaric spinal anesthetics and cerebrospinal fluid movement. Anesth Analg. 2011;112:924-30.*

29. *Greene NM, Brull SJ. Physiology of Spinal Anesthesia, 4th ed. Baltimore: Williams & Wilkins. 1993: 85-199, 309-43, and 357-80*

30. *Stoelting's pharmacology and physiology in anaesthetic practice 5th edition;chapter-18;pg 679-684*

31. *Stoelting's pharmacology and physiology in anaesthetic practice 5th edition;chapter-18;pg 688-692*

32. *Cabalum 77, Zugaib M, Lieb S, Nuwayhid B, Brinkman CR III, Assali NS. Effects of dopamine on hypotension in- duced by spinal anesthesia. Am J Obstet Gynecol 1979; 133: 630-4. 41*

33. *Clark RB, Brunner JA III. Dopamine for the treatment of spinal hypotension during cesarean section. Anesthesiology 1980; 53: 514-7. 42*

34. *Butterworth JF IV, Piccione W Jr, Berrizbeitia LD, Dance G, Shemin R J, Cohn LH. Augmentation of venous return of adrenergic agonists during spinal anesthesia. Anesth Analg 1986; 65: 612-6. 49*

35. *Lundberg J, Norgren L, Thomson D, Werner 0 Hemodynamic effects of dopamine during thoracic epidural analgesia in man. Anesthesiology 1987; 66: 641-6.*

36. *Kasaba T, Yamaga M, Iwasaki T et al: Ephedrine, dopamine, or dobutamine to treat hypotension with propofol during epidural anesthesia. Can J Anaesth, 2000; 47(3): 237–41*

37. *Lundberg J, Lundberg D, Norgren L, Werner O: Dopamine counteracts hypertension during general anesthesia and hypotension during combined thoracic epidural anesthesia for abdominal aortic surgery. J Cardiothorac Anesth, 1990; 4(3): 348–53*

38. *Rolbin SH, Levinson G, Shnider SM, Biehl DR, Wright RG.Anesthesiology. 1979 Jul;51(1):37-40.*

39. *Effects of Continuous Intravenous Infusion of Methoxamine on the Intraoperative Hemodynamics of Elderly Patients Undergoing Total Hip Arthroplasty http://www.medscimonit.com/abstract/index/idArt/890760*

40. *Ward R J, Kennedy WF Jr, Bonica J J,, Martin WE, Tolas AG, Akamatsu T. Experimental evaluation of atropine and vasopressors for the treatment of hypotension of high sub- arachnoid anesthesia. Anesth Analg 1966; 45: 621-9.*

41. *Butterworth JF IV, Piccione W Jr, Berrizbeitia LD, Dance G, Shemin R J, Cohn LH. Augmentation of venous return of adrenergic agonists during spinal anesthesia. Anesth Analg 1986; 65: 612-6.*

42. *Prophylactic Phenylephrine Infusion for Preventing Hypotension During Spinal Anesthesia for Cesarean Delivery.Ngan Kee, Warwick D. MBChB, MD, FANZCA; Khaw, Kim S. MBBS, FRCA; Ng, Floria F. RN, BASc; Lee, Bee B. MBBS, FANZCA*

43. *Frazer RS Edwards GM. Prophylactic ephedrine infusion in obstetric anaesthesia (Letter). Br J Anaesth 1990; 64: 651*

44. *Kang YG, Abouleish E, Caritis S. Prophylactic intravenous ephedrine infusion during spinal anesthesia for cesarean section. Anesth Analg 1982; 61: 839–42*

45. *Gajraj NM, Victory RA, Pace NA, Van Elstraete AC, Wallace DH. Comparison of an ephedrine infusion with crystalloid administration for prevention of hypotension during spinal anesthesia. Anesth Analg 1993; 76: 1023–6*

46. *Hemmingsen C, Poulsen JA, Risbo A. Prophylactic ephedrine during spinal anaesthesia: double-blind study in patients in ASA groups I–III. Br J Anaesth 1989; 63: 340–2.*

47. *Techniques for preventing hypotension during spinal anaesthesia for caesarean section,Allan M Cyna[1,*], Marion Andrew[2], Richard S Emmett[2], Philippa Middleton[3], Scott W Simmons[4] http://onlinelibrary.wiley.com/o/cochrane/clabout/articles/ oct 2006*

48. *Coe AJ, Revanäs B. Forum: Is crystalloid preloading useful in spinal anaesthesia in the elderly? Anaesthesia. 1990; 45:241.*

49. *Engberg G, Wiklund L. The circulatory effects of intravenously administered ephedrine during epidural blockade. Acta Anaesthesiol Scand, 1978 Suppl; 66: 27–36.*

50. *Prevention of hypotension during spinal anaesthesia for Caesarean section: ephedrine infusion versus fluid preload.W. S. Chan[1], M. G. Irwin[1], W. N. Tong[1,†]and Y. H. Lam[2.] .BJA VOL-52,ISSUE 9,pg 908-913,sep-1997.*

51. *R.Jackson et al, volume preloading is not essential to prevent spinal induced hypotension in caesarian section. Bja-1995,75,pg 262-265*

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ANNEXURES

INSTITUTIONAL ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No.19/03/2015 Meeting held on 26/03/2015
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Randomised control study of various effects of intravenous dopamine continuous infusion on intraoperative haemodynamics in patient undergoing elective surgeries under spinal anaesthesia" – For Dissertation Purpose" submitted by Dr.J.Saravanan, MD (Anaesthesia), Govt. Kilpauk Medical College, Chennai - 10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

31/03/15

CHAIRMAN,
Ethical Committee
Govt. Kilpauk Medical College, Chennai

31/03/15

Comp<E Colon< MEI Sec<Ethical Committee 1

PROFORMA

Name:

Age:

Sex:

IPno:
Ward/ SU

Group:

Date of admission:
Date of surgery:

ASA Physical Status:

Co- Morbidity:

Patient on any drugs:

Preoperative examination:

Blood pressure:

Pulse rate :

Room air SpO2:

Cardiovascular system:

Respiratory system:

Central nervous system:

Diagnosis:

Surgery being performed:

Investigations:

Premedication:

Time of injection of study drug:

Group (Tick any one)

- Group-A: patients who do not receive dopamine infusion
- Group-B: patients who receive dopamine infusion@3mcg/kg/mt
- Group-C:patients who receive dopamine infusion@5mcg/kg/mt
- Group-D:Patients who receive dopamineinfusion@7mcg/kg/mt

Duration of surgery :

Position during surgery:

OBSERVATIONS:

INTRAOPERATIVE PARAMETERS:

TIME	SBP	DBP	MAP	HR	SPO2	URINE OUTPUT
1 Min. after SAB¶						
5 Min. after SAB. ¶						
10Min. after SAB. ¶						
15 Min. after SAB¶.						
20 Min. after SAB. ¶						
25 Min. after SAB. ¶						

30 Min. after SAB. ¶						
40 Min. after SAB¶						
50 Min. after SAB. ¶						
60 Min. after SAB. ¶						
70 Min. after SAB. ¶						
80 Min. after SAB. ¶						
90 min after SAB						
100 min afer SAB						
110 min after SAB						
120 min after SAB						

PATIENT CONSENT FORM

Study detail : A PROSPECTIVE RANDOMISED CONTROL STUDY OF EFFECTS OF VARIOUS DOSES OF DOPAMINE CONTINUOUS INTRAVENOUS INFUSION ON THE INTRAOPERATIVE HEMODYNAMICS IN PATIENTS UNDERGOING ELECTIVE SURGERIES UNDER SPINAL ANAESTHESIA

Study Centre : GOVT. KILPAUK MEDICAL COLLEGE HOSPITAL & GOVT. ROYAPETTAH HOSPITAL, CHENNAI.

Patients Name :

Patients Age :

Identification Number :

Patient may check (✓) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb impression:

Patients Name and Address: place date

Signature of investigator :

Study investigator's Name : place date

நோயாளி ஒப்புதல் படிவம்

ஆராய்ச்சியின் விவரம் :

ஆராய்ச்சி மையம் : அரசு கீழ்பாக்கம் மருத்துவக் கல்லூரி மருத்துவமனை

நோயாளியின் பெயர் : நோயாளியின் வயது:

பதிவு எண் :

நோயாளி கீழ்கண்டவற்றுள் கட்டங்களை (✓) செய்யவும்

1 மேற்குறிப்பிட்டுள்ள ஆராய்ச்சியின் நோக்கத்தையும் பயனையும் முழுவதுமாக புரிந்து கொண்டேன். மேலும் எனது அனைத்து சந்தேங்களையும் கேட்டு அதற்கான விளக்கங்களையும் தெளிவுபடுத்திக் கொண்டேன். ☐

2 மேலும் இந்த ஆராய்ச்சிக்கு எனது சொந்த விருப்பத்தின் பேரில் பங்கேற்கிநேன் என்றும், மேலும் எந்த நேரத்திலும் எவ்வித முன்றிவிப்பு மின்றி இந்த ஆராய்ச்சியிலிருந்து விலக முழுமையான உரிமை உள்ளதையும் இதற்கு எவ்வித சட்ட பிணைப்பும் இல்லை என்பதையும் அறிவேன். ☐

3 ஆராய்சியாளரோ, ஆராய்ச்சி உதவியாளரோ, ஆராய்ச்சி உபயத்தாரரோ, ஆராய்ச்சி பேராசிரியரோ, ஒழுங்குநெறி செயற்குழு உறுப்பினர்களோ எப்போது வேண்டுமானாலும் எனது அனுமதியின்றி எனது உள்நோயாளி மற்றும் புற நோயாளி பதிவுகளை இந்த ஆராய்ச்சிக்காகவோ அல்லது எதிர்கால பிறஆராய்ச்சிகளுக்காகவோ பயன்படுத்திக் கொள்ளலாம் என்றும் மேலும் இந்த நிபந்தனை நான் இவ்வராய்ச்சிலிருந்து தகும் என்றும் ஒப்புக்கொள்கிறேன். ஆயினும் எனது அடையாளம் சம்பந்தப்பட்ட எந்த பதிவகளும் (சட்டபூர்வமான தேவைகள் தவிர) வெளியிடப்படமாட்டது என்ற உறுதிமொழியின் பெயரில் இந்த ஆராய்ச்சிலிருந்து கிடைக்கப்பெறும் முடிவுகளை வெளியிட மறுப்பு தெறிவிக்கமாட்டேன் என்று உறுதியளிக்கிறேன். ☐

4. இந்த ஆராய்ச்சிஆச்ன வாயின் அருகில் வரும் சீழ் கட்டியை குறித்தது. அந்த நோயின் தன்மையையும், பின் விளைவுகளையும் பற்றியும், அறுவை சிகிச்சையின் போது கீறி எடுக்கப்படும் சீழை பரிசோதனைக்கு அனுப்பி கிருமியின் தன்மையையும் அதற்கு உகந்த மருந்தை பற்றியும் அறிய நடத்தும் ஆராய்ச்சி என்பதை மருத்துவர் மூலம் அறிந்து கொண்டேன். ☐
5. இந்த ஆராய்ச்சிக்கு நான் முழுமனதுடன் சம்மதிக்கின்றேன் என்றும் மேலும் ஆராய்ச்சி குழுவினர் எனக்கு அளிக்கும் அறிவுரைகளை தவறாது பின்பற்றுவேன் என்றும் உறுதியளிக்கிறேன். ☐
6. இந்த ஆராய்ச்சிக்குத் தேவைப்படும் அனைத்து மருத்துவப்பரிசோதனைகளுக்கும் ஒத்துழைப்பு தருவேன் என்று உறுதியளிக்கிறேன். ☐
7. இந்த ஆராய்ச்சிக்கு யாருடைய எற்புறுத்தலுமின்றி எனது சொந்த விருப்பத்தின் பேரிலும் சுயஅறிவுடனும் முழுமனதுடனும் சம்மதிக்கின்றேன் என்று இதன் மூலம் ஒப்புக்கொள்கிறேன். ☐

நோயாளியின் கையொப்பம் / பெருவிரல் கைரேகை
இடம்: தேதி:

ஆராய்ச்சியாளரின் கையொப்பம்:
இடம்: தேதி:

Master chart group A art group A

name	age	sex	asa status	height	weight	Diagnosis	procedure	preop sbp	preop dbp	preop map	preop hr	sbp1	dbp1	map1	hr1
anzad basha	33	m	ps1	165	56	RIH	hernioplasty	134	88	103	68	122	85	97	68
kannan	38	m	ps1	170	70	B/LIH	hernioplasty	140	80	101	86	133	86	98	76
leon josephine	49	m	ps1	158	52	LIH	hernioplasty	120	78	96	90	122	75	95	94
gangammal	55	f	ps2	155	56	IN.H	hernioplasty	157	105	122	106	109	71	84	118
aarumugam	64	m	ps2	165	58	RIH	hernioplasty	124	90	101	78	110	70	88	76
ramachandran	65	m	ps2	162	50	LIH	hernioplasty	155	82	106	68	130	74	93	76
loganathan	40	m	ps1	165	70	RIH	hernioplasty	122	80	90	80	119	78	88	82
chellammal	60	f	ps2	152	56	RIH	hernioplasty	150	80	104	95	140	86	98	90
premkumar	25	m	ps1	164	62	RIH	hernioplasty	107	65	79	93	113	79	90	88
suresh	40	m	ps1	168	58	RIH	hernioplasty	121	82	95	88	119	82	92	106
kannaiah	43	m	ps2	157	53	LIH	hernioplasty	150	90	108	88	120	89	99	86
raj	45	m	ps1	165	66	LIH	hernioplasty	147	94	109	80	133	76	94	76
antony raj	38	m	ps1	164	58	RIH	hernioplasty	120	68	96	64	119	68	93	63
velmurugan	40	m	ps1	170	70	LIH	hernioplasty	156	104	120	110	148	95	104	103
parasuraman	34	m	ps1	158	55	LIH	hernioplasty	150	70	120	90	149	91	108	88
engel shaw	27	m	ps1	170	70	RIH	hernioplasty	133	88	101	68	122	87	92	61
pencillaiah	55	m	ps2	156	45	LIH	hernioplasty	133	88	101	84	101	76	86	86
akbar	64	m	ps1	162	55	RIH	hernioplasty	142	86	104	88	136	72	98	86
veerasamy	65	m	ps2	168	58	LIH	hernioplasty	146	84	98	64	117	60	78	60
vijaya	43	f	ps2	155	60	UH	hernioplasty	142	85	104	95	109	65	80	92
ayyappan	62	m	ps2	158	54	LIH	hernioplasty	134	86	101	64	110	72	78	61
sanny	60	m	ps2	170	60	IN.H	hernioplasty	150	86	106	102	147	89	107	108
bakthavatchalam	64	m	ps2	168	65	RIH	hernioplasty	150	88	107	94	140	90	100	88
rajkumar	48	m	ps2	165	58	RIH	hernioplasty	128	90	101	80	124	89	100	78
chandran	63	p	ps2	162	50	LIH	hernioplasty	154	84	104	70	155	82	106	68
kumar	25	m	ps1	168	62	RIH	hernioplasty	114	80	92	94	107	65	79	93
barathan	64	m	ps1	166	58	RIH	hernioplasty	142	86	104	88	136	72	98	86
paramesh	34	m	ps1	158	55	LIH	hernioplasty	150	70	120	90	149	91	108	88
rajasekar	45	m	ps1	165	66	LIH	hernioplasty	149	96	109	82	147	94	109	80
surendar	40	m	ps1	168	68	RIH	hernioplasty	128	86	98	84	121	82	95	88

sbp2	dbp2	map2	hr2	sbp3	dbp3	map3	hr3	sbp4	dbp4	map4	hr4	sbp5	dbp5	map5	hr5
121	88	97	71	96	69	78	60	101	70	80	59	101	70	80	59
130	84	97	77	128	85	96	78	132	84	97	74	133	87	100	72
116	74	82	93	112	74	81	91	112	76	81	92	111	72	86	91
92	59	70	110	85	56	66	99	90	60	70	105	89	61	70	106
106	73	84	76	104	75	85	66	105	75	85	77	93	75	81	52
69	41	50	51	85	51	62	53	85	51	62	56	89	52	64	57
118	77	87	76	112	73	81	74	112	73	81	76	113	72	82	69
135	85	92	88	128	72	88	86	108	68	76	83	99	61	70	80
98	65	76	89	98	65	76	86	99	66	77	69	98	64	75	72
116	72	90	102	114	74	92	98	109	68	76	82	109	67	76	72
90	61	70	78	84	57	68	80	108	72	76	85	103	71	78	78
99	63	73	69	106	65	78	69	98	60	74	66	115	70	85	68
127	68	97	61	117	62	88	62	110	59	72	60	112	60	81	63
131	88	98	102	127	84	90	98	133	98	104	98	130	88	100	92
150	88	106	100	150	85	104	100	150	90	108	104	141	85	102	88
120	80	90	59	118	78	89	61	119	80	90	64	122	83	92	62
88	57	67	89	103	67	78	88	101	66	77	76	90	60	68	75
124	74	96	84	110	72	92	84	104	68	84	80	99	63	74	78
89	51	63	61	97	56	67	63	97	56	67	57	100	52	64	58
85	49	61	70	103	69	80	96	89	59	69	90	105	72	83	98
98	65	75	62	104	72	79	61	115	78	87	63	119	80	90	64
140	87	101	107	147	84	103	105	149	92	110	109	129	83	95	108
117	77	86	69	111	67	90	87	103	63	76	81	110	68	85	82
110	72	89	76	106	73	84	76	104	75	85	66	105	75	85	77
130	74	93	76	69	41	50	51	85	51	62	53	85	51	62	56
113	79	90	88	98	65	76	89	98	65	76	86	99	66	77	69
124	74	96	84	110	72	92	84	104	68	84	80	99	63	74	78
150	88	106	100	150	85	104	100	150	90	108	104	141	85	102	88
133	76	94	76	99	63	73	69	106	65	78	69	98	60	74	66
119	82	92	106	116	72	90	102	114	74	92	98	109	68	76	82
sbp10	dbp10	map10	hr10	sbp15	dbp15	map15	hr15	sbp20	dbp20	map20	hr20	sbp25	dbp25	map25	hr25

101	71	82	59	101	72	82	59	101	72	82	58	101	72	82	58
129	84	95	78	128	84	95	72	129	84	95	69	132	84	97	64
115	69	86	85	107	64	74	81	104	65	72	77	99	63	70	79
87	61	70	102	109	69	82	104	102	68	79	99	100	60	73	81
114	78	90	56	119	86	97	82	112	82	92	78	102	76	85	77
87	52	64	58	105	59	74	57	104	58	73	58	101	57	71	56
111	73	81	69	111	72	81	71	113	72	82	70	108	65	78	73
89	58	68	80	102	72	78	78	110	63	80	78	96	58	66	62
97	63	74	67	92	57	69	60	92	56	68	58	92	55	67	58
95	61	76	72	94	64	72	67	96	59	67	67	98	67	90	66
96	65	74	78	99	71	75	74	110	79	84	74	104	74	82	72
106	62	78	69	95	59	68	65	103	62	72	66	96	59	73	62
116	50	71	58	109	54	81	58	113	56	72	57	104	54	75	58
124	84	95	88	121	80	91	78	121	81	91	76	129	85	97	84
138	80	96	76	136	79	94	77	137	85	96	77	134	81	92	77
130	83	96	65	133	90	102	61	133	89	102	65	130	87	100	63
97	64	74	75	86	57	69	72	98	76	82	74	84	56	62	70
119	66	81	80	103	53	75	82	112	65	78	68	108	66	78	66
98	52	66	58	99	52	64	58	102	55	68	54	103	56	71	56
104	70	81	97	102	67	79	90	100	66	77	91	100	66	77	88
127	86	96	62	130	84	96	61	119	81	89	59	127	84	95	60
119	80	89	108	121	81	91	102	107	68	78	103	118	79	88	95
132	84	90	76	120	76	86	60	119	75	86	58	119	73	84	57
93	75	81	52	114	78	90	56	119	86	97	82	112	82	92	78
89	52	64	57	87	52	64	58	105	59	74	57	104	58	73	58
98	64	75	72	97	63	74	67	92	57	69	60	92	56	68	58
119	66	81	80	103	53	75	82	112	65	78	68	108	66	78	66
138	80	96	76	136	79	94	77	137	85	96	77	134	81	92	77
115	70	85	68	106	62	78	69	95	59	68	65	103	62	72	66
109	67	76	72	95	61	76	72	94	64	72	67	96	59	67	67

sbp30	dbp30	map30	hr30	sbp40	dbp40	map40	hr40	sbp50	dbp50	map50	hr50	sb60	dbp60	map60	hr60	u/o 60
100	72	81	59	99	72	81	58	106	77	87	70	98	74	82	58	75
140	89	104	70	129	85	97	63	121	84	79	62	130	84	96	62	100
97	56	67	78	100	56	67	71	99	59	68	72	105	62	79	71	50
99	60	73	90	92	57	69	81	97	57	70	85	94	59	71	92	50
98	73	81	77	93	68	76	62	100	76	84	76	95	73	80	77	50
100	57	71	55	109	57	76	60	110	68	80	66	109	68	78	69	100
115	75	84	70	130	81	93	71	123	82	92	83	130	83	96	82	50
107	61	74	64	108	59	76	67	105	55	70	64	108	60	78	70	75
91	55	67	55	91	53	66	55	94	56	69	58	94	57	69	59	0
97	60	75	65	97	62	75	65	102	63	77	60	106	69	86	60	100
118	80	89	70	111	76	82	68	115	80	90	68	117	80	89	64	50
105	63	77	62	99	60	71	62	105	63	73	63	113	64	87	58	75
106	60	76	56	107	60	80	55	105	49	74	56	102	51	76	53	100
129	85	97	85	131	85	99	71	147	88	104	69	147	92	108	71	100
135	83	94	73	135	81	99	70	132	80	98	71	132	90	101	70	50
133	88	101	67	129	87	99	60	140	93	107	61	132	89	101	61	50
101	71	78	70	96	65	74	69	99	67	81	68	96	66	75	63	50
110	68	76	69	106	64	74	70	110	68	78	68	114	69	78	72	50
109	58	73	55	111	60	76	54	112	62	78	55	112	62	78	53	100
99	65	76	84	97	62	74	83	106	72	83	89	106	70	82	83	75
122	81	90	58	127	80	93	61	120	82	91	61	128	85	96	57	75
106	73	81	82	96	64	74	97	128	79	92	92	124	81	92	92	100
118	72	83	60	119	74	85	59	120	71	84	58	123	74	86	64	75
102	76	85	77	98	73	81	77	93	68	76	62	100	76	84	76	100
101	57	71	56	100	57	71	55	109	57	76	60	110	68	80	66	100
92	55	67	58	91	55	67	55	91	53	66	55	94	56	69	58	0
110	68	76	69	106	64	74	70	110	68	78	68	114	69	78	72	50
135	83	94	73	135	81	99	70	132	80	98	71	132	90	101	70	50
96	59	73	62	105	63	77	62	99	60	71	62	105	63	73	63	75
98	67	90	66	97	60	75	65	97	62	75	65	102	63	77	60	100

sbp70	dbp70	map70	hr70	sbp80	dbp80	map80	hr80	sbp90	dbp90	map90	hr90	sbp100	dbp100	map100	hr100
111	77	88	60	109	74	86	60	106	72	83	59				
123	83	94	66	129	89	100	60								
100	56	69	69	99	60	76	74	100	70	82	74				
95	60	72	78	100	64	76	85	110	67	81	88				
96	73	81	80	97	74	82	77	97	75	82	71	100	78	85	80
129	83	95	78												
121	66	82	67	109	60	81	63	113	62	84	64	121	65	88	72
94	57	69	58	101	60	74	62	100	60	73	58	112	78	82	62
116	68	86	60	115	74	86	60	112	73	94	60	122	74	100	64
119	83	90	64	120	85	92	66								
112	65	91	62	109	72	90	60	119	72	102	63	129	74	90	64
107	51	73	54	106	53	69	58	102	53	68	52	104	48	66	54
144	92	107	78	143	95	109	78	142	94	108	78	144	96	111	80
131	91	102	60	131	87	100	56	140	91	105	56	134	90	103	58
93	65	73	68	98	66	76	64	102	70	78	60	106	72	80	62
118	72	80	68	122	78	82	69								
111	61	77	57	120	64	79	52	117	62	78	53	119	64	78	56
106	70	82	84	107	70	82	79	105	70	82	82	107	72	84	84
134	86	101	61	124	84	96	71								
114	78	86	94	108	68	78	95	113	71	82	77	111	74	83	89
119	72	83	59	133	78	92	62	121	77	88	62	127	75	88	66
95	73	80	77	96	73	81	80	97	74	82	77	97	75	82	71
109	68	78	69												
94	57	69	59	94	57	69	58	101	60	74	62	100	60	73	58
118	72	80	68	122	78	82	69								
113	64	87	58	112	65	91	62	109	72	90	60	119	72	102	63
106	69	86	60	116	68	86	60	115	74	86	60	112	73	94	60

Sbp 110	Dbp 110	Map 110	Hr 110	Sbp 120	Dbp 120	Map 120	hr120	u/o 120	ephedrine	ephedrine dose(mg)	atropine	SPO2<95	Sens.bloc level
									Y	18	N	N	T8
									N	0	N	N	T8
									Y	18	N	N	T8
									Y	24	N	N	T6
102	78	86	77					150	Y	6	N	N	T8
									Y	18	N	N	T8
									N	0	N	N	T10
									Y	18	N	N	T6
									Y	12	N	N	T6
									Y	12	N	N	T8
									Y	18	N	N	T8
									Y	24	N	N	T6
107	49	72	56					150	Y	18	N	N	T8
									N	0	N	N	T10
									N	0	N	N	T10
									N	0	N	N	T10
									Y	30	N	N	T8
									Y	18	N	N	T8
117	62	78	55	115	63	79	51	200	Y	18	N	N	T8
108	72	84	78	109	72	84	78	150	Y	18	N	N	T6
									Y	6	N	N	T8
117	77	86	76	122	80	90	81		Y	12	N	N	T8
122	78	88	61						Y	12	N	N	T8
100	78	85	80	102	78	86	77	150	Y	6	N	N	T8
									Y	18	N	N	T8
112	78	82	62						Y	12	N	N	T6
									Y	18	N	N	T8
									N	0	N	N	T10
129	74	90	64						Y	24	N	N	T6

Master chart group B

Name	age	sex	asa status	ht	wt	Diag	procedure	preop sbp	preop dbp	preop map	preop hr	sbp1	dbp1	map1
thangamani	47	m	ps1	158	54	LIH	hernioplasty	128	76	98	78	119	76	91
Chinnaiah	62	m	ps2	160	58	LIH	hernioplasty	142	81	98	88	124	77	89
usman basha	25	m	ps1	166	60	RIH	hernioplasty	134	80	98	98	129	82	98
Rajagopal	52	m	ps2	164	50	RIH	hernioplasty	119	81	90	102	119	77	88
Rajesh	40	m	ps1	155	57	LIH	hernioplasty	134	78	100	82	128	76	98
Kiran	60	m	ps2	168	62	RIH	hernioplasty	135	84	99	96	134	80	98
Jagannath	25	m	ps1	165	54	RIH	hernioplasty	124	80	90	100	118	78	89
Rekha	40	f	ps2	153	55	UH	hernioplasty	130	81	101	80	118	80	90
umesh kumar	43	m	ps1	167	66	RIH	hernioplasty	136	80	100	98	130	84	98
Soorya	45	m	ps2	160	59	RIH	hernioplasty	120	82	91	88	108	78	88
Anzad	38	m	ps1	152	64	LIH	hernioplasty	128	76	98	78	119	76	91
Ibrahim	40	m	ps2	163	70	RIH	hernioplasty	134	80	98	98	129	82	98
Mohan	34	m	ps1	168	63	RIH	hernioplasty	121	80	90	102	119	77	88
Shalab	27	m	ps1	156	58	LIH	hernioplasty	128	76	98	78	119	76	91
parameswari	55	f	ps2	159	65	IN.H	hernioplasty	134	80	98	98	129	82	98
Saravanan	64	m	ps2	161	63	RIH	hernioplasty	119	81	90	102	119	77	88
Ramesh	65	m	ps1	166	54	LIH	hernioplasty	128	76	98	78	119	76	91
Santhanam	43	m	ps1	162	66	RIH	hernioplasty	134	80	98	98	129	82	98
Krishnan	62	m	ps2	165	56	RIH	hernioplasty	119	81	90	102	119	77	88
barath kumar	60	m	ps1	157	55	LIH	hernioplasty	128	76	98	78	119	76	91
Madhusudanan	64	m	ps2	166	61	RIH	hernioplasty	134	80	98	98	129	82	98
Latha	48	f	ps1	170	53	UH	hernioplasty	119	81	90	102	119	77	88
nambirajan	63	m	ps1	157	48	LIH	hernioplasty	128	76	98	78	119	76	91
ilaya murugan	25	m	ps1	169	60	RIH	hernioplasty	134	80	98	98	129	82	98
Nagendran	64	m	ps2	165	54	RIH	hernioplasty	119	81	90	102	119	77	88
Rajendran	34	m	ps1	150	42	LIH	hernioplasty	128	76	98	78	119	76	91
Sivakumar	45	m	ps1	161	60	RIH	hernioplasty	142	83	118	88	132	71	92
jayaprakash	40	m	ps1	165	66	RIH	hernioplasty	119	81	90	102	119	77	88
Magesh	45	m	ps1	155	55	LIH	hernioplasty	128	76	98	78	119	76	91
muthukumar	48	m	ps2	164	58	RIH	hernioplasty	123	83	93	97	119	81	90

sbp2	dbp2	map2	hr2	sbp3	dbp3	map3	hr3	sbp4	dbp4	map4	hr4	sbp5	dbp5	map5	hr5
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
103	63	76	86	91	58	67	90	92	59	68	85	86	52	63	71
128	82	97	84	127	82	97	83	126	82	97	99	125	84	98	84
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
119	76	91	78	110	72	81	84	109	65	79	81	107	67	79	83
129	82	98	89	128	82	97	84	127	82	97	83	126	82	97	99
114	64	79	99	98	61	72	98	112	58	70	96	92	52	64	101
109	70	80	82	108	63	78	80	106	66	78	81	99	64	72	80
126	80	96	82	124	80	95	81	122	82	97	99	125	84	98	84
104	72	84	86	102	70	80	84	103	58	70	82	92	52	65	85
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
128	82	97	84	127	82	97	83	126	82	97	99	125	84	98	84
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
128	82	97	84	127	82	97	83	126	82	97	99	125	84	98	84
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
128	82	97	84	127	82	97	83	126	82	97	99	125	84	98	84
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
128	82	97	84	127	82	97	83	126	82	97	99	125	84	98	84
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
128	82	97	84	127	82	97	83	126	82	97	99	125	84	98	84
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
137	64	89	91	137	64	95	89	133	59	94	91	132	55	86	83
104	64	76	98	99	60	71	96	102	56	69	98	93	53	63	100
110	72	81	84	109	65	79	81	107	67	79	83	97	62	73	81
118	80	89	91	119	78	88	90	117	76	85	84	120	77	87	82

sbp10	dbp10	map10	hr10	sbp15	dbp15	map15	hr15	sbp20	dbp20	map20	hr20	sbp25	dbp25	map25	hr25
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
109	67	79	78	126	67	82	78	144	75	100	75	146	80	100	78
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	62	73	81	97	59	71	77	100	63	74	77	98	62	73	69
125	84	98	84	125	83	97	86	124	92	108	69	124	84	97	62
89	58	66	106	101	63	75	96	105	62	76	95	103	65	77	94
96	58	70	78	101	64	74	76	99	61	74	68	100	62	74	70
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
86	56	62	78	100	60	74	78	106	68	80	74	110	68	81	76
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
125	83	97	86	124	92	108	69	124	84	97	62	122	75	91	60
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
137	64	95	74	130	67	92	72	132	65	86	71	131	66	95	74
88	57	64	102	102	63	75	96	105	62	76	95	103	65	77	94
97	59	71	77	100	63	74	77	98	62	73	69	99	61	73	68
111	66	79	83	109	67	78	81	111	73	82	70	112	72	81	81

sbp30	dbp30	map30	hr30	sbp40	dbp40	map40	hr40	sbp50	dbp50	map50	hr50	sb60	dbp60	map60	hr60	u/o 60
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	1000
134	73	90	81	122	72	85	77	124	71	86	77	129	73	88	70	700
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	500
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	450
99	61	73	68	101	62	74	67	101	64	75	68	103	64	76	67	650
122	75	91	60	126	79	95	61	125	80	95	65	120	81	94	63	550
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	450
101	64	75	69	102	65	76	69	102	65	75	69	105	70	79	74	800
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	500
122	75	91	60	126	79	95	61	125	80	95	65	120	81	94	63	600
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	900
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	500
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	450
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	400
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	900
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	450
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	700
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	500
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	450
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	800
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	500
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	750
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	650
126	79	95	61	125	80	95	65	120	81	94	63	122	81	95	63	500
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	450
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	700
133	63	83	85	140	60	91	82	143	67	93	76	140	56	94	72	500
109	63	78	95	115	67	81	100	120	72	84	93	113	65	78	96	550
101	62	74	67	101	64	75	68	103	64	76	67	104	69	78	69	800
113	76	84	81	121	80	90	86	118	73	84	84	120	73	85	73	400

sbp70	dbp70	map70	hr70	sbp80	dbp80	map80	hr80	sbp90	dbp90	map90	hr90	sbp100	dbp100	map100	hr100
104	63	76	64	111	67	78	70	109	71	81	70				
133	75	91	73	133	74	90	77	133	80	95	75				
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	69	78	69	104	63	76	64	111	67	78	70	109	71	81	70
122	81	95	63	130	87	101	74	130	81	97	62	128	81	97	66
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
106	64	78	65	114	68	79	68	110	70	80	72	111	72	82	74
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92				
104	63	76	64	111	67	78	70	109	71	81	70				
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	63	76	64	111	67	78	70	109	71	81	70				
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	63	76	64	111	67	78	70	109	71	81	70				
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	63	76	64	111	67	78	70	109	71	81	70				
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	63	76	64	111	67	78	70	109	71	81	70				
130	87	101	74	130	81	97	62	128	81	97	66	124	88	100	58
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	63	76	64	111	67	78	70	109	71	81	70				
148	56	96	71	139	67	94	77	133	70	94	78				
119	60	78	96	118	71	83	97	120	71	83	92	121	72	84	91
104	63	76	64	111	67	78	70	109	71	81	70				
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77

ephedrine	ephedrine dose(mg)	atropine	SPO2<95	sensory blockade level
N	0	N	N	T8
Y	6	N	N	T8
N	0	N	N	T8
Y	6	N	N	T8
N	0	N	N	T8
N	0	N	N	T8
Y	12	N	N	T6
N	0	N	N	T8
N	0	N	N	T10
Y	12	N	N	T6
N	0	N	N	T8
N	0	N	N	T8
Y	12	N	N	T6
N	0	N	N	T8
N	0	N	N	T8
Y	18	N	N	T6
N	0	N	N	T8
N	0	N	N	T8
Y	6	N	N	T8
N	0	N	N	T8
N	0	N	N	T8
Y	6	N	N	T10
N	0	N	N	T8
N	0	N	N	T10
Y	6	N	N	T8
N	0	N	N	T8
N	0	N	N	T10
Y	6	N	N	T6
N	0	N	N	T8
N	0	N	N	T10

Master chart group C

name	Age	sex	asa status	ht	wt	Diag	procedure	preop sbp	preop dbp	preop map	preop hr	sbp1	dbp1	map1	hr1
muthukumar	25	m	ps1	170	68	RIH	hernioplasty	132	83	118	88	132	71	92	92
neelagandan	55	m	ps2	165	50	RIH	hernioplasty	119	80	93	68	109	63	78	72
annamalai	57	m	ps2	162	50	UH	hernioplasty	138	76	98	79	130	85	96	88
krishnakumar	51	m	ps2	164	62	RIH	hernioplasty	123	83	93	97	119	81	90	88
Sundar	25	m	ps1	170	65	RIH	hernioplasty	124	83	97	85	120	83	95	73
murugesan	65	m	ps2	159	55	RIH	hernioplasty	142	88	104	86	133	88	101	89
ayyammal	48	f	ps1	155	58	LIH	hernioplasty	130	90	102	90	145	98	112	96
perumal	43	m	ps1	155	68	RIH	hernioplasty	142	83	118	88	132	71	92	92
gandhi	62	m	ps2	158	50	RIH	hernioplasty	109	80	93	68	109	63	78	72
murugan	60	m	ps2	170	50	UH	hernioplasty	138	76	98	79	130	85	96	88
kathiravan	64	m	ps2	168	62	RIH	hernioplasty	123	83	93	97	119	81	90	88
manikandan	48	m	ps1	165	65	RIH	hernioplasty	124	83	97	85	120	83	95	73
sivaraj	63	m	ps2	162	55	RIH	hernioplasty	132	88	104	86	133	88	101	89
sirajul islam	25	m	ps1	168	68	RIH	hernioplasty	132	83	118	88	132	71	92	92
pandurangan	64	m	ps2	166	50	RIH	hernioplasty	129	80	93	68	109	63	78	72
paulraj	34	m	ps2	158	50	UH	hernioplasty	138	76	98	79	130	85	96	88
dinavel	45	m	ps2	165	62	RIH	hernioplasty	134	84	95	92	123	83	93	97
gopinath	40	m	ps1	168	65	RIH	hernioplasty	124	83	97	85	120	83	95	73
moses	65	m	ps2	159	55	RIH	hernioplasty	132	88	104	86	133	88	101	89
sudha	48	f	ps1	155	58	LIH	hernioplasty	130	90	102	90	145	98	112	96
venkat sai	25	m	ps1	170	68	RIH	hernioplasty	132	83	118	88	132	71	92	92
masilamani	55	m	ps2	165	50	RIH	hernioplasty	119	80	93	68	109	63	78	72
chandra bose	57	m	ps2	162	50	UH	hernioplasty	138	76	98	79	130	85	96	88
nagamani	51	m	ps2	164	62	RIH	hernioplasty	123	83	93	97	119	81	90	88
rajasekar	25	m	ps1	170	65	RIH	hernioplasty	124	83	97	85	120	83	95	73
ezhumalai	65	m	ps2	159	55	RIH	hernioplasty	142	88	104	86	133	88	101	89
kanimozhi	48	f	ps1	155	58	LIH	hernioplasty	140	90	102	90	145	98	112	96
santhosh	25	m	ps1	170	68	RIH	hernioplasty	132	83	118	88	132	71	92	92
baskar	57	m	ps2	162	50	UH	hernioplasty	138	76	98	79	130	85	96	88
latha	48	f	ps1	155	58	LIH	hernioplasty	140	90	102	90	145	98	112	96

sbp2	dbp2	map2	hr2	sbp3	dbp3	map3	hr3	sbp4	dbp4	map4	hr4	sbp5	dbp5	map5	hr5
137	64	89	91	137	64	95	89	133	59	94	91	132	55	86	83
115	75	88	73	108	70	83	68	106	69	87	67	104	69	87	66
129	70	88	99	152	85	100	103	146	78	99	106	142	72	98	92
118	80	89	91	119	78	88	90	117	76	85	84	120	77	87	82
122	84	97	81	122	85	97	77	123	82	96	67	124	82	96	68
129	85	97	83	142	88	103	79	141	83	100	81	133	84	99	86
152	92	110	81	148	92	110	79	148	90	109	82	143	89	105	82
137	64	89	91	137	64	95	89	133	59	94	91	132	55	86	83
115	75	88	73	108	70	83	68	106	69	87	67	104	69	87	66
129	70	88	99	152	85	100	103	146	78	99	106	142	72	98	92
118	80	89	91	119	78	88	90	117	76	85	84	120	77	87	82
122	84	97	81	122	85	97	77	123	82	96	67	124	82	96	68
129	85	97	83	142	88	103	79	141	83	100	81	133	84	99	86
137	64	89	91	137	64	95	89	133	59	94	91	132	55	86	83
115	75	88	73	108	70	83	68	106	69	87	67	104	69	87	66
129	70	88	99	152	85	100	103	146	78	99	106	142	72	98	92
119	81	90	88	118	80	89	91	119	78	88	90	117	76	85	84
122	84	97	81	122	85	97	77	123	82	96	67	124	82	96	68
129	85	97	83	142	88	103	79	141	83	100	81	133	84	99	86
152	92	110	81	148	92	110	79	148	90	109	82	143	89	105	82
137	64	89	91	137	64	95	89	133	59	94	91	132	55	86	83
115	75	88	73	108	70	83	68	106	69	87	67	104	69	87	66
129	70	88	99	152	85	100	103	146	78	99	106	142	72	98	92
118	80	89	91	119	78	88	90	117	76	85	84	120	77	87	82
122	84	97	81	122	85	97	77	123	82	96	67	124	82	96	68
129	85	97	83	142	88	103	79	141	83	100	81	133	84	99	86
152	92	110	81	148	92	110	79	148	90	109	82	143	89	105	82
137	64	89	91	137	64	95	89	133	59	94	91	132	55	86	83
129	70	88	99	152	85	100	103	146	78	99	106	142	72	98	92
152	92	110	81	148	92	110	79	148	90	109	82	143	89	105	82

sbp10	dbp10	map10	hr10	sbp15	dbp15	map15	hr15	sbp20	dbp20	map20	hr20	sbp25	dbp25	map25	hr25
137	64	95	74	130	67	92	72	132	65	86	71	131	66	95	74
104	67	79	63	103	67	79	66	107	65	77	60	100	65	77	60
133	69	98	105	130	64	88	97	132	70	90	99	127	60	88	81
111	66	79	83	109	67	78	81	111	73	82	70	112	72	81	81
122	82	95	71	121	82	95	77	122	81	95	66	121	81	94	65
131	86	99	78	128	81	94	75	127	81	93	69	142	84	102	67
147	89	107	80	141	86	103	79	140	87	102	78	143	92	108	77
137	64	95	74	130	67	92	72	132	65	86	71	131	66	95	74
104	67	79	63	103	67	79	66	107	65	77	60	100	65	77	60
133	69	98	105	130	64	88	97	132	70	90	99	127	60	88	81
111	66	79	83	109	67	78	81	111	73	82	70	112	72	81	81
122	82	95	71	121	82	95	77	122	81	95	66	121	81	94	65
131	86	99	78	128	81	94	75	127	81	93	69	142	84	102	67
137	64	95	74	130	67	92	72	132	65	86	71	131	66	95	74
104	67	79	63	103	67	79	66	107	65	77	60	100	65	77	60
133	69	98	105	130	64	88	97	132	70	90	99	127	60	88	81
120	77	87	82	111	66	79	83	109	67	78	81	111	73	82	70
122	82	95	71	121	82	95	77	122	81	95	66	121	81	94	65
131	86	99	78	128	81	94	75	127	81	93	69	142	84	102	67
147	89	107	80	141	86	103	79	140	87	102	78	143	92	108	77
137	64	95	74	130	67	92	72	132	65	86	71	131	66	95	74
104	67	79	63	103	67	79	66	107	65	77	60	100	65	77	60
133	69	98	105	130	64	88	97	132	70	90	99	127	60	88	81
111	66	79	83	109	67	78	81	111	73	82	70	112	72	81	81
122	82	95	71	121	82	95	77	122	81	95	66	121	81	94	65
131	86	99	78	128	81	94	75	127	81	93	69	142	84	102	67
147	89	107	80	141	86	103	79	140	87	102	78	143	92	108	77
137	64	95	74	130	67	92	72	132	65	86	71	131	66	95	74
133	69	98	105	130	64	88	97	132	70	90	99	127	60	88	81
147	89	107	80	141	86	103	79	140	87	102	78	143	92	108	77

sbp30	dbp30	map30	hr30	sbp40	dbp40	map40	hr40	sbp50	dbp50	map50	hr50	sb60	dbp60	map60	hr60	u/o 60
133	63	83	85	140	60	91	82	143	67	93	76	140	56	94	72	500
100	65	77	57	100	66	77	59	102	67	77	65	105	68	80	69	1000
111	56	82	79	122	56	82	79	125	59	88	76	130	75	100	88	500
113	76	84	81	121	80	90	86	118	73	84	84	120	73	85	73	400
120	81	94	67	123	79	94	74	123	77	92	60	119	77	91	62	800
128	83	94	69	133	82	96	70	132	80	94	72	133	81	94	70	650
145	87	104	78	131	86	99	76	141	85	102	76	141	85	100	76	1100
133	63	83	85	140	60	91	82	143	67	93	76	140	56	94	72	500
100	65	77	57	100	66	77	59	102	67	77	65	105	68	80	69	1000
111	56	82	79	122	56	82	79	125	59	88	76	130	75	100	88	500
113	76	84	81	121	80	90	86	118	73	84	84	120	73	85	73	400
120	81	94	67	123	79	94	74	123	77	92	60	119	77	91	62	800
128	83	94	69	133	82	96	70	132	80	94	72	133	81	94	70	900
133	63	83	85	140	60	91	82	143	67	93	76	140	56	94	72	500
100	65	77	57	100	66	77	59	102	67	77	65	105	68	80	69	1300
111	56	82	79	122	56	82	79	125	59	88	76	130	75	100	88	500
112	72	81	81	113	76	84	81	121	80	90	86	118	73	84	84	450
120	81	94	67	123	79	94	74	123	77	92	60	119	77	91	62	750
128	83	94	69	133	82	96	70	132	80	94	72	133	81	94	70	950
145	87	104	78	131	86	99	76	141	85	102	76	141	85	100	76	1000
133	63	83	85	140	60	91	82	143	67	93	76	140	56	94	72	550
100	65	77	57	100	66	77	59	102	67	77	65	105	68	80	69	700
111	56	82	79	122	56	82	79	125	59	88	76	130	75	100	88	550
113	76	84	81	121	80	90	86	118	73	84	84	120	73	85	73	450
120	81	94	67	123	79	94	74	123	77	92	60	119	77	91	62	850
128	83	94	69	133	82	96	70	132	80	94	72	133	81	94	70	900
145	87	104	78	131	86	99	76	141	85	102	76	141	85	100	76	1200
133	63	83	85	140	60	91	82	143	67	93	76	140	56	94	72	600
111	56	82	79	122	56	82	79	125	59	88	76	130	75	100	88	400
145	87	104	78	131	86	99	76	141	85	102	76	141	85	100	76	1100

sbp70	dbp70	map70	hr70	sbp80	dbp80	map80	hr80	sbp90	dbp90	map90	hr90	sbp100	dbp100	map100	hr100
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
136	78	98	74	147	89	107	78	134	80	96	74				
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
120	73	85	73	111	73	82	75	112	75	83	79	119	76	89	75
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
136	78	98	74	147	89	107	78	134	80	96	74				
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
136	78	98	74	147	89	107	78	134	80	96	74				
148	56	96	71	139	67	94	77	133	70	94	78				
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
136	78	98	74	147	89	107	78	134	80	96	74				

sbp70	dbp70	map70	hr70	sbp80	dbp80	map80	hr80	sbp90	dbp90	map90	hr90	sbp100	dbp100	map100	hr100
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
136	78	98	74	147	89	107	78	134	80	96	74				
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
120	73	85	73	111	73	82	75	112	75	83	79	119	76	89	75
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
136	78	98	74	147	89	107	78	134	80	96	74				
148	56	96	71	139	67	94	77	133	70	94	78				
108	70	80	70	112	78	88	78								
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
111	73	82	75	112	75	83	79	119	76	89	75	122	81	91	77
108	72	84	63	109	72	84	66	110	71	84	66	109	73	85	68
128	81	92	74	121	78	89	72	121	83	109	76	127	84	108	74
136	78	98	74	147	89	107	78	134	80	96	74				
148	56	96	71	139	67	94	77	133	70	94	78				
127	66	84	80	129	78	96	85	131	74	96	84	130	77	98	85
136	78	98	74	147	89	107	78	134	80	96	74				

ephedrine	ephedrine dose(mg)	atropine	SPO2<95	sensory blockade level
N	0	N	N	T8
N	0	N	N	T8
N	0	N	N	T10
N	0	N	N	T8
N	0	N	N	T8
N	0	N	N	T6
N	0	N	N	T8
N	0	N	N	T10
N	0	N	N	T8
N	0	N	N	T6
N	0	N	N	T6
N	0	N	N	T8
N	0	N	N	T6
N	0	N	N	T6
N	0	N	N	T8
N	0	N	N	T10
N	0	N	N	T6
N	0	N	N	T8
N	0	N	N	T8
N	0	N	N	T6
N	0	N	N	T10
Y	6	N	N	T8
N	0	N	N	T8
N	0	N	N	T8
N	0	N	N	T8
N	0	N	N	T8
N	0	N	N	T6
N	0	N	N	T6
N	0	N	N	T8
N	0	N	N	T8

Master chart group D

name	age	sex	asa status	height	weight	diagnosis	procedure	preop sbp	preop dbp	preop	preop hr	sbp1	dbp1	map1	hr1
muthukumar	25	m	ps1	170	68	RIH	hernioplas	142	83	118	88	132	71	92	92
neelagandan	55	m	ps2	165	50	rt inguinal	hernioplas	119	80	93	68	109	63	78	72
annamalai	57	m	ps2	162	50	umblical	hernioplas	148	76	98	79	130	85	96	88
krishnakumar	51	m	ps2	164	62	rt inguinal	hernioplas	123	83	93	97	119	81	90	88
Sundar	25	m	ps1	170	65	rt inguinal	hernioplas	124	83	97	85	120	83	95	73
murugesan	65	m	ps2	159	55	rt inguinal	hernioplas	142	88	104	86	133	88	101	89
ayyammal	48	f	ps1	155	58	lt inguinal	hernioplas	140	90	102	90	145	98	112	96
perumal	43	m	ps1	155	68	rt inguinal	hernioplas	142	83	118	88	132	71	92	92
gandhi	62	m	ps2	158	50	rt inguinal	hernioplas	109	80	93	68	109	63	78	72
murugan	60	m	ps2	170	50	umblical	hernioplas	148	76	98	79	130	85	96	88
kathiravan	64	m	ps2	168	62	rt inguinal	hernioplas	123	83	93	97	119	81	90	88
manikandan	48	m	ps1	165	65	rt inguinal	hernioplas	124	83	97	85	120	83	95	73
sivaraj	63	m	ps2	162	55	rt inguinal	hernioplas	142	88	104	86	133	88	101	89
sirajul islam	25	m	ps1	168	68	rt inguinal	hernioplas	142	83	118	88	132	71	92	92
pandurangan	64	m	ps2	166	50	rt inguinal	hernioplas	129	80	93	68	109	63	78	72
paulraj	34	m	ps2	158	50	umblical	hernioplas	148	76	98	79	130	85	96	88
dinavel	45	m	ps2	165	62	rt inguinal	hernioplas	134	84	95	92	123	83	93	97
gopinath	40	m	ps1	168	65	rt inguinal	hernioplas	124	83	97	85	120	83	95	73
moses	65	m	ps2	159	55	rt inguinal	hernioplas	142	88	104	86	133	88	101	89
sudha	48	f	ps1	155	58	lt inguinal	hernioplas	140	90	102	90	145	98	112	96
venkat sai	25	m	ps1	170	68	rt inguinal	hernioplas	142	83	118	88	132	71	92	92
masilamani	55	m	ps2	165	50	rt inguinal	hernioplas	119	80	93	68	109	63	78	72
chandra bose	57	m	ps2	162	50	umblical	hernioplas	148	76	98	79	130	85	96	88
nagamani	51	m	ps2	164	62	rt inguinal	hernioplas	123	83	93	97	119	81	90	88
rajasekar	25	m	ps1	170	65	rt inguinal	hernioplas	124	83	97	85	120	83	95	73
ezhumalai	65	m	ps2	159	55	rt inguinal	hernioplas	142	88	104	86	133	88	101	89
kanimozhi	48	f	ps1	155	58	lt inguinal	inguinal	140	90	102	90	145	98	112	96
santhosh	25	m	ps1	170	68	rt inguinal	hernioplas	142	83	118	88	132	71	92	92
baskar	57	m	ps2	162	50	umblical	hernioplas	148	76	98	79	130	85	96	88
latha	48	f	ps1	155	58	lt inguinal	hernioplas	140	90	102	90	145	98	112	96

sbp2	dbp2	map2	hr2	sbp3	dbp3	map3	hr3	sbp4	dbp4	map4	hr4	sbp5	dbp5	map5	hr5
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
111	85	90	80	106	74	98	80	120	79	89	75	112	77	84	73
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
142	91	105	74	149	93	111	86	150	94	111	82	159	93	115	74
115	76	90	58	121	78	100	62	134	81	98	60	130	68	88	60
96	46	64	78	95	53	68	74	94	54	67	86	98	48	63	84
106	74	98	80	120	79	89	75	112	77	84	73	111	72	81	73

sbp10	dbp10	map10	hr10	sbp15	dbp15	map15	hr15	sbp20	dbp20	map20	hr20	sbp25	dbp25	map25	hr25
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
111	72	81	73	110	71	81	70	105	68	78	66	108	67	78	72
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
156	89	109	68	145	87	105	67	145	84	102	65	161	87	110	62
134	78	99	61	146	84	111	58	155	88	122	60	166	89	119	60
104	50	68	88	107	50	69	88	105	52	68	85	109	56	75	80
110	71	81	70	105	68	78	66	108	67	78	72	118	65	80	77

sbp30	dbp30	map30	hr30	sbp40	dbp40	map40	hr40	sbp50	dbp50	map50	hr50	sb60	dbp60	map60	hr60	u/o 60
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	1200
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	450
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	850
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	550
118	65	80	77	114	75	83	76	115	75	83	76	118	74	85	67	1000
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	400
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	500
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	800
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	1050
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	450
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	550
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	450
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	1300
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	850
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	550
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	450
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	650
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	850
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	550
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	450
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	900
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	850
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	450
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	350
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	1000
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	850
152	83	105	70	144	79	97	65	147	84	104	67	131	82	95	74	650
159	89	120	61	144	76	103	59	136	72	98	60	134	86	99	61	950
113	52	76	84	114	55	68	80	114	53	70	86	119	59	77	85	550
114	75	83	76	115	75	83	76	118	74	85	67	121	72	84	78	1100

sbp70	dbp70	map70	hr70	sbp80	dbp80	map80	hr80	sbp90	dbp90	map90	hr90	sbp100	dbp100	map100	hr100
122	72	85	73	121	79	89	69	117	75	85	69	114	81	88	72
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
121	78	93	60	123	76	94	58	120	74	84	76	116	69	85	74
127	77	87	69	129	81	93	73	120	75	87	71	122	74	86	76
121	72	84	78	122	72	85	73	121	79	89	69	117	75	85	69
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
127	77	87	69	129	81	93	73	120	75	87	71	122	74	86	76
121	78	93	60	123	76	94	58	120	74	84	76	116	69	85	74
122	72	85	73	121	79	89	69	117	75	85	69	114	81	88	72
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
127	77	87	69	129	81	93	73	120	75	87	71	122	74	86	76
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
122	72	85	73	121	79	89	69	117	75	85	69	114	81	88	72
121	78	93	60	123	76	94	58	120	74	84	76	116	69	85	74
127	77	87	69	129	81	93	73	120	75	87	71	122	74	86	76
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
122	72	85	73	121	79	89	69	117	75	85	69	114	81	88	72
121	78	93	60	123	76	94	58	120	74	84	76	116	69	85	74
127	77	87	69	129	81	93	73	120	75	87	71	122	74	86	76
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
122	72	85	73	121	79	89	69	117	75	85	69	114	81	88	72
121	78	93	60	123	76	94	58	120	74	84	76	116	69	85	74
127	77	87	69	129	81	93	73	120	75	87	71	122	74	86	76
113	59	77	87	120	61	80	90	123	60	84	75	126	70	87	94
122	72	85	73	121	79	89	69	117	75	85	69	114	81	88	72

ephedrine	ephedrine	atropine	SPO2<95	sensory	step downto 5 mcg/kg/mt	time of stepdown(mt)
N	0	N	N	T6	N	
N	0	N	N	T8	N	
N	0	N	N	T10	Y	25
N	0	N	N	T8	Y	25
N	0	N	N	T8	N	
N	0	N	N	T6	N	
N	0	N	N	T8	Y	20
N	0	N	N	T8	Y	25
N	0	N	N	T8	N	
N	0	N	N	T6	N	
N	0	N	N	T8	Y	30
N	0	N	N	T8	N	
N	0	N	N	T8	N	
N	0	N	N	T10	Y	25
N	0	N	N	T8	Y	20
N	0	N	N	T6	N	
N	0	N	N	T6	N	
N	0	N	N	T10	Y	25
N	0	N	N	T8	Y	30
N	0	N	N	T6	N	
N	0	N	N	T6	N	
N	0	N	N	T8	Y	25
N	0	N	N	T8	Y	25
N	0	N	N	T8	N	
N	0	N	N	T8	N	
N	0	N	N	T10	Y	20
N	0	N	N	T8	Y	20
N	0	N	N	T10	Y	25
N	0	N	N	T6	N	
N	0	N	N	T8	N	